**Summary Minutes of the CASAC Particulate Matter (PM)
Review Panel Teleconference Consultation on EPA's Risk Analysis Plans for
Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}**

May 1, 2003, Ariel Rios Building, Washington, D.C.

Panel members:	See Roster (Attachment A)	
Date and Time:	10:00 A.M. to 12:00 P.M. EDT, May 1, 2003	
Location:	U.S. EPA, Ariel Rios Building North, Conference Room 6013, 1200 Pennsylvania Avenue, N.W., Washington, DC 20004	
Purpose:	The purpose of this meeting was for the CASAC Particulate Matter (PM) Review Panel to conduct a consultation with EPA on Risk Analysis Plans for Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}.	
Attendees:	Chair:	Dr. Philip Hopke
	CASAC Members:	Dr. Frederick Miller Mr. Richard Poirot Dr. Frank Speizer Dr. Sverre Vedal Dr. Barbara Zielinska
	Consultants:	Dr. Jane Koenig Dr Petros Koutrakis Dr. Allan Legge Dr. Paul J. Lioy Dr. Morton Lippmann Dr. Joe Mauderly Dr. Roger McClellan Dr. Gunter Oberdorster Dr. Robert Rowe Mr. Ronald White Dr. Warren White Dr. George Wolff
	EPA SAB Staff:	Mr. Fred Butterfield, DFO Mr. Bob Flaak

Other attendees:

OAQPS Staff	Dr. Karen Martin Mr. John Bachmann Mr. Harvey Richmond Dr. Mary Ross Mr. John Langstaff Ms. Linda Chappell Ms. Lisa Conner
ORD Staff	Dr. Lester Grant Dr. William Wilson Mr. Bob Fegley Dr. Rob Elias Mr. Bill Russo Ms. Barbara Glenn
OGC Staff	Mr. Steve Silverman Mr. Gerald Gleason
Abt Associates (EPA contractor)	Dr. Leland Deck Dr. Ellen Post Mr. Etienne Gabel
Public	John Huess, Air Pollutant Resource Kurt Blaze, O'Connor and Hammond Cliff Michaelson, NESCAUM Courtney Shrum, American Lung Association (ALA) Will Ollison, Dick Carp, Kyle Isakower, API Bill Johnson, NESCAUM Bruce Hill, Clean Air Taskforce Laura Bibbs, North Carolina Madge Pritts, New York State DOH Lisa Hershberger, Minnesota Pollution Control Agency Steve Cook, BNA Teri Pierce, Consolidated Safety Services

Meeting Summary

The discussion generally followed the issues and timing as presented in the meeting agenda (Attachment B), although there were no public comments. The meeting lasted until 11:55 a.m.

Introductions and Administration

Mr. Fred Butterfield, Designated Federal Officer (DFO) for the Committee, opened the meeting and asked participants to introduce themselves, as well as to state their names for the record before speaking. He expressed appreciation to panel members, the public, and the staff in Research Triangle Park for their participation. He briefly reviewed the meeting agenda, confirmed that there were no members of the public who wanted to comment during the teleconference, and noted that one set of written public comments had been received to date.

Purpose of Meeting

Dr. Philip Hopke, chair of the Clean Air Scientific Advisory Committee (CASAC) and CASAC PM Review Panel, welcomed participants and summarized the purpose of this consultation meeting (*Federal Register* Notice, Attachment C). Part of the input materials for the PM Staff Paper is a risk assessment, which will be conducted to assess the degree of health risk posed by current PM concentrations. The results of the assessment will be incorporated into the Staff Paper. Today's consultation meeting will provide input into how the Office of Air Quality Planning and Standards (OAQPS) is going to proceed and give insights into potential problems with developing plans for the risk assessment.

Highlights of EPA's Risk Analysis Plans for Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}

Mr. Richmond thanked Dr. Hopke and the panel, and noted that representatives of the contractor, Abt Associates, Inc., were present. He began his presentation on the plans for PM health risk analyses for coarse PM and PM_{10} by providing background information on previous PM NAAQS review (1996/1997) and the role the CASAC has served in developing the current draft plans for PM coarse ($PM_{10-2.5}$) and PM_{10} risk analyses (Slides, Attachment D). Mr. Richmond noted that CASAC had previously agreed that a consultation meeting should be held on plans for $PM_{10-2.5}$ risk analyses. The revised Staff Paper will be produced later this summer. Mr. Richmond explained that the general methodology for $PM_{10-2.5}$ and PM_{10} is the same as described in the January 2002 report for $PM_{2.5}$. He noted that OAQPS did not plan to analyze risk for alternative PM_{10} standards. The proposed scope of the health risk analyses was presented, which includes the selection of health endpoint categories, urban areas, epidemiological studies, and concentration-response relationships to include in the analyses.

Mr. Richmond indicated that the selection of health endpoints would focus on the more severe and better-understood endpoints, and where the weight of evidence, based on the evaluation in the PM criteria document (CD), supports a likely causal relationship. For PM_{10}, mortality, hospital admissions (and possibly ER visits), and respiratory symptoms would be examined. For $PM_{10-2.5}$, hospital admissions (and possibly ER visits), and respiratory symptoms would be examined. Mr. Richmond noted that the city of Pittsburgh had been added to those urban areas to be included in the PM_{10} risk analysis.

A general discussion of city selection, particularly on including other urban areas in the analyses, followed. The criterion of "C-R functions with greater statistical power" was of particular interest to several panelists. In response to a panelist's question about cutoff points and use of the NMMAPS study in the draft of the staff paper, Mr. Richmond noted that the authors of the NMMAPS study urged caution in projecting city-specific results outside the top 20 cities. Dr. Ross indicated that the inclusion of additional urban areas was based on an indicator of statistical power based on the log of the product of the number of days and health endpoints in a study using 9 as a rough cutpoint based on total mortality. Dr. Grant further explained that a plot of the NMMAPS cities and health endpoints had been done, and there was a narrowing of the confidence interval beyond the natural log of total mortality-days greater than or equal to 9. A review panelist suggested that given the variability that can occur in coarse particles, this should be the absolute minimum level. Others suggested somewhat relaxing the restriction to see if more studies can be included in Exhibit 1 of the 4/8/03 draft Abt memo (Attachment E).

Another review panelist questioned the lack of cities with a high proportion of minorities and whether there were sufficient data to add cities such as Birmingham and Atlanta. A general discussion ensued in which several high minority-population cities were mentioned for inclusion, such as Atlanta, Nashville, Birmingham, New Orleans, and New York. Studies mentioned included ARIES, Lipfert's study of Philadelphia, and a study by Gwynn and Thurston (2001) that looked at hospital admissions by race in New York City. It was noted that Detroit is a city with a large black population and that it was included in the planned risk analyses. Concerns about geographic coverage were also expressed.

The issue of PM_{10} analysis was raised, and concern was expressed by a panelist about a possible point of diminishing returns considering the amount of PM_{10} analysis being done when the EPA is not considering alternative PM_{10} standards. Another panelist spoke in favor of PM_{10} analysis, and emphasized that the strongest analyses are those where all three indicators are available, rather than focusing on one indicator and perhaps missing the big picture. Mr. Richmond emphasized that it was the intention of OAQPS to consider as many of the three indicators (i.e., $PM_{2.5,}$ PM_{10}, and $PM_{10-2.5}$) as are available and cited Detroit and Phoenix as examples where studies using all three indicators were available.

Mr. Richmond presented the criteria for the selection of studies to be analyzed. First, they must be acceptable, published, peer-reviewed studies evaluated in the draft PM CD. Second, PM must be directly measured using PM_{10} or $PM_{10-2.5}$ as indicators. Third, only concentration-response (C-R) relationships from studies not involving the GAM/S-Plus issue and from peer-reviewed re-analyses of GAM studies will be used. Mr. Richmond further explained the proposed approach for selecting C-R relationships. He noted that there are a number of locations where there are studies using multiple indicators.

There was a general discussion of various studies and the reasons for their exclusion from the proposed risk analysis. Seattle was mentioned, and Mr. Richmond noted that it pertained to only PM_{10}, not $PM_{10-2.5}$. In response to a question, Dr. Ross reported that 75-80 percent of the data are missing for fine particles in the Shepherd study, so it was not included. Phoenix, Detroit and Atlanta were discussed; one panelist expressed his belief that whatever data are available from ARIES should be used for Atlanta if they have been described in the peer reviewed literature.

A panelist mentioned that additional studies in Steubenville and Phoenix have reported statistically significant effects associated with $PM_{10-2.5.}$ Dr. Wilson noted two major differences from the other cities. First, soil contamination by metals was present. Second, rather small areas were sampled (a very small portion of the Phoenix area was examined) and one would not expect to see much. The negatives are not evidence that coarse PM is not toxic; but rather you don't have the ability to see them in that type of situation.

Panelists noted that $PM_{10-2.5}$ particles were more heterogeneous than $PM_{2.5}$ particles and were not as well measured relative to $PM_{2.5}$. Several panelists expressed support for including acute mortality for $PM_{10-2.5}$ among the health endpoints to be included in the risk assessment and the CASAC chair summarized that it was the view of the Committee to include mortality for $PM_{10-2.5.}$ A panelist emphasized his belief that it is important to recognize the spectrum of health endpoints and how they shift for different indicators in the analysis and the interpretation of the analysis. Another panelist suggested that until the various re-analyses are completed and reviewed by HEI, EPA staff, and the Committee, it is unclear where the weight of evidence lies for any of the PM metrics. Mr. Richmond indicated a willingness to move forward with using mortality data for $PM_{10-2.5}$ with appropriate caveats.

Mr. Richmond reviewed the amended table of health endpoints and urban locations for short-term exposure (Table 1 in Attachment D) and the proposed approach for selecting C-R relationships. Both single and multi-pollutant functions will be included where available. For reanalyzed GAM studies, the GAM C-R function with more stringent convergence criteria will be used. Lag(s) recommended by authors of the studies or the draft PM CD will be used, and distributed lag will be included.. A panelist suggested that David Chock's analysis of PM in Pittsburgh should be added to the PM_{10} assessment because he not only used a GLM but also has seasonal results for the seasonal analysis. Mr. Richmond noted that Dr. Chock's Pittsburgh study is not currently listed but will be included for total mortality and the NMMAPS reanalyzed

hospital admission study which included Pittsburgh also will be included.. A panelist commented that because most PM_{10} coefficients in the re-analysis of Samet *et al*. 2000 for total non-accidental mortality are not significant, the lower confidence limit for these C-R functions will be negative.

In response to a panelist's question, Mr. Richmond said that the Chicago and Los Angeles studies referenced in Exhibit 3 of the preliminary re-analysis document (Attachment E) are not restricted to a single author, but rather, multiple studies by different authors are used. A panelist raised an issue relative to the NMMAPS study; its purpose is to estimate regional or national effects so that when considering individual cities, one has to qualify it somewhat in that the investigators haven't optimally modeled each individual city. It was remarked that the authors might argue that they have a bit less confidence in the mortality data because the cities are not individually modeled, whereas for the morbidity data the individual cities were modeled with different specifications.

One review panelist expressed strong support for the use of multiple models as proposed for Chicago and Los Angeles, noting that NMMAPS looks broadly at variation among cities and regional differences.

A panelist suggested that there are so few data on asthma (morbidity) that the big picture may be missed if EPA excudes the study in Seattle where $PM_{2.5}$ data were filled in with data measured using nepholometers. Mr. Richmond asked the air quality members of the panel for comments on the acceptability of using this study based on nepholometry data. The panel expressed support for using studies based on nephelometry data with an appropriate footnote, as those data are a good indicator of fine particle mass.

A lengthy discussion of the issue of background and how to correct for it ensued. Some panelists stated that trying to quantify background level is extremely difficult and not worth the effort for purposes of the risk assessment. Other panelists expressed caution that the effect of PM would be overestimated if background was not considered, and that there is an obligation to try to separate it out.

It was noted that there are contributions to background from a variety of natural processes as well as uncontrollable processes (e.g., wind-blown dust from agricultural land, wildfires, activities outside the U.S., and biological material). Mr. Bachmann provided some historical perspective, cautioning that there were strong policy reasons for EPA to consider only risks in excess of background in setting NAAQS and this has been EPA policy for a long time.

In this context, there was discussion among panelists about the purpose of risk assessment and whether its primary purpose is to provide administrators with information about the portion of PM that is controlable. The question was raised of how to properly separate the natural contribution to PM levels from what can be controlled.

The CASAC chair summarized by noting that we have struggled many years with defining background and how to estimate it and suggested that a majority of the panel agreed that it is probably better and more scientifically defensible to calculate the risk based on the original data and then apportion it between controllable and non-controllable so that one averages these risks and then subtracts the risk associated with a constant, average background. This decreases the influence of high background events like dust storms.

Mr. Richmond stated that the various points on background would be taken into consideration by the staff.

Dr. Hopke asked if there were any other issues the panel wished to discuss. A review panelist asked about respiratory hospital admissions and ER visits as a health endpoint, questioning whether it was a sound approach to estimate the ratio between ER visits and hospital admissions. Mr. Richmond stated that there are some studies on the ratio of ER visits to hospital admissions, and since national survey data are available, a ratio approach could be used for the baseline. The difficulty of obtaining accurate ER data representative for an urban area was discussed, with the variability of rates across both regions and within a given urban area particularly noted by several panelists. One panelist suggested that ER visit data could be obtained by directly contacting hospitals. Another panelist expressed concern that all hospitals in an area would need to be contacted or there might be bias in the rates obtained.

Dr. Hopke asked that any additional comments be written up and sent to Mr. Butterfield. He reminded panel members that since this is a consultation, no consensus report will be produced. Mr. Richmond thanked all the members of the panel for their insights and participation.

Dr. Hopke asked Dr. Grant for a timeline for the remainder of the project. Dr. Grant stated his expectation that the criteria document would be ready the last week in June, followed by a 60-day public comment period. A CASAC PM Review Panel meeting will be held the last week in August and the Staff Paper will be released at the end of August. An additional meeting on the Staff Paper will be held before the end of the calendar year.

Mr. Butterfield expressed his appreciation to the members of the panel and the public. He asked the panel members to pencil in Tuesday August 26-Wednesday August 27 as the most likely dates for the meeting, which will be held in Research Triangle Park. Another meeting will be held in November to review the draft PM Staff Paper and risk assessment, sometime before Thanksgiving. Mr. Butterfield will canvass the committee in a post-meeting e-mail to determine when the largest quorum can be present for both meetings, so that the dates can be selected as

soon as possible.

Dr. Hopke adjourned the meeting at 11:55 a.m.

ATTACHMENTS

Attachment A: Roster of the CASAC
Attachment B: Meeting Agenda
Attachment C: *Federal Register* Notice
Attachment D: OAQPS Presentation
Attachment E: April 8, 2003 Draft Abt Associates Memo
Attachment F: Public Comments: Utility Air Regulatory Group
Attachment G: Public Comments: Coalition for Coarse Particle Regulation

Attachment A

U.S. Environmental Protection Agency
Science Advisory Board
Clean Air Scientific Advisory Committee
CASAC Particulate Matter Review Panel*

CHAIR

Dr. Philip Hopke, Bayard D. Clarkson Distinguished Professor, Department of Chemical Engineering, Clarkson University, Potsdam, NY

Also Member: Research Strategies Advisory Committee
Executive Committee

CASAC MEMBERS

Dr. Frederick J. Miller, Vice President for Research, CIIT Centers for Health Research, Research Triangle Park, NC

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. George E. Taylor, Professor and Assistant Dean, School of Computational Sciences, George Mason University, Fairfax, VA

Dr. Sverre Vedal, Professor of Medicine, National Jewish Medical and Research Center, Denver, CO, USA

Dr. Barbara Zielinska, Research Professor , Division of Atmospheric Science, Desert Research Institute, Reno, NV

CONSULTANTS

Dr. Jane Q. Koenig, Professor, Department of Environmental Health, School of Puublic Health and Community Medicine, University of Washington, Seattle, WA

Dr. Petros Koutrakis, Professor, Environmental Science and Engineering Program, School of Public Health, Harvard University, Boston, MA

Dr. Allan Legge, President, Biosphere Solutions, Calgary, Alberta, CANADA

Dr. Paul J. Lioy, Associate Director and Professor, Environmental and Occupational Health Sciences Institute, UMDNJ - Robert Wood Johnson Medical School, Piscataway, NJ

Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Joe Mauderly, Vice President, Senior Scientist, and Director, National Environmental Respiratory Center, Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Roger O. McClellan, Consultant, Albuquerque, NM

Dr. Gunter Oberdorster, Professor of Toxicology, Department of Environmental Medicine, University of Rochester, Rochester, NY

Dr. Robert D. Rowe, President, Stratus Consulting, Inc., Boulder, CO

Dr. Jonathan M. Samet, Professor and Chair, Department of Epidemiology, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD

Mr. Ronald White, Assistant Executive Director, National Osteoporosis Foundation, Silver Spring, MD

Dr. Warren H. White, Senior Research Associate , Chemistry Department, Washington University, St. Louis, MO

Dr. George T. Wolff, Principal Scientist, General Motors Corporation, Detroit, MI

SCIENCE ADVISORY BOARD STAFF

Mr. Fred Butterfield, Designated Federal Officer, 1200 Pennsylvania Avenue, NW, Washington, DC, Phone: 202-564-4561, Fax: 202-501-0582, (butterfield.fred@epa.gov)

Delores Darden , Office Assistant, 1200 Pennsylvania Avenue, NW, Washington, DC, Phone: 202-564-2282, Fax: 202-501-0582, (darden.delores@epa.gov)

* Members of this SAB Panel consist of

a. SAB Members: Experts appointed by the Administrator to serve on one of the SAB Standing Committees.

b. SAB Consultants: Experts appointed by the SAB Staff Director to a one-year term to serve on ad hoc Panels formed to address a particular issue.

c. Liaisons: Members of other Federal Advisory Committees who are not Members or Consultants of the Board.

d. Federal Experts: "Federal Experts" are federal employees who have technical knowledge and expertise relevant to the subject matter under review or study by a particular panel.

Attachment B

U.S. Environmental Protection Agency
Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel

Thursday, May 1, 2003 – Public Teleconference Meeting
10:00 am - 12:00 pm Eastern Time
Ariel Rios Federal Building North – Conference Room 6013
1200 Pennsylvania Avenue, NW, Washington, DC 20460

Teleconference Consultation on EPA's Risk Analysis Plans for Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}

Final Meeting Agenda

Thursday, May 1, 2003

10:00 am	**Convene Teleconference; Call Attendance Introductions and Administration**	Mr. Fred Butterfield, CASAC DFO
10:10 am	**Purpose of Meeting**	Dr. Phil Hopke, Chair
10:15 am	**Highlights of EPA's Risk Analysis Plans for Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}, with discussion by CASAC Members**	Mr. Harvey Richmond, OAQPS Health & Ecosystems Effects Group
10:30 am	**CASAC Members' Discussion**	Dr. Hopke and CASAC
11:25 pm	**Public Comment Period**	Mr. Butterfield (Facilitator)
11:45 pm	**Summary and Next Steps**	Dr. Hopke
12:00 pm	**Adjourn Meeting** (time approximate)	Mr. Butterfield

The survey will target systems in two categories: systems which have had violations of one or more chosen rulemakings and systems which have not had violations (but have made compliance decisions to prevent a violation). An initial short survey will be used to identify a sample of systems that have made compliance decisions in response to the representative rulemakings without actually having been out of compliance. The full survey (including a pilot study phase) will be sent to these systems, as well as to a sample of systems that have recorded violations. We estimate that the initial survey (known as a screener survey, since it will identify respondents appropriate for the full survey) will provide data from 1,875 respondents indicating whether or not they made some type of compliance decision associated with the representative rulemakings. We estimate that the full survey (including a pilot study phase), sent to systems with and without recorded violations, will provide data from 718 respondents.

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR part 9 and 48 CFR chapter 15.

The EPA would like to solicit comments to:

(i) Evaluate whether the proposed collection of information is necessary for the proper performance of the functions of the Agency, including whether the information will have practical utility;

(ii) Evaluate the accuracy of the Agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used;

(iii) Enhance the quality, utility, and clarity of the information to be collected; and

(iv) Minimize the burden of the collection of information on those who are to respond, via the use of appropriate automated electronic, mechanical, or other technological collection techniques or other forms of information technology, *e.g.*, permitting electronic submission of responses.

III. What Are EPA's Burden and Cost Estimates for This ICR?

The following is a summary of the burden and cost estimates associated with this proposed information collection effort. Burden and cost estimates are taken from the ICR, which provides a detailed explanation of the burden estimates summarized in this notice. EPA anticipates that the only entities affected by this information request will be public water systems. The total number of estimated potential respondents is 1,875 for the screener survey and 718 for the full survey. Respondents to the screener survey will only have to respond to that survey once. Respondents to the full survey will only have to respond to the full survey once. Some respondents, however, will have to respond to both the screener survey and the full survey. EPA estimates that 1,567 respondents will respond once to the screener survey, 410 respondents will respond once to the full survey, and 308 respondents will respond once to both the screener survey and the full survey.

The annual public burden for this collection of information is estimated to average 0.25 hours per screener survey response; 1 hour per full survey response for small public water systems; 2 hours per full survey response for medium public water systems; and 3 hours per full survey response for large public water systems. The estimated total annual respondent burden for screener survey respondents is 469 hours with a current annual cost of $10,742; the estimated total annual respondent burden for full survey respondents is 1,304 hours with a current annual cost of $34,204. Total estimated annual respondent burden associated with the complete information collection effort is 1,773 hours with a current annual cost of $44,946.

Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

Dated: March 21, 2003.

Cynthia C. Dougherty,

Director, Office of Ground Water and Drinking Water.

[FR Doc. 03–9046 Filed 4–11–03; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[FRL –7482–8]

Science Advisory Board, Clean Air Scientific Advisory Committee, Notification of Public Advisory Committee Meeting; Teleconference Consultation on Risk Analysis Plans for Coarse Particulate Matter ($PM_{10–2.5}$) and PM_{10}

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The Environmental Protection Agency (EPA), Science Advisory Board (SAB), announces the conduct of a publically-accessible teleconference of the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel to review the Agency's risk analysis plans for coarse-fraction $PM_{10–2.5}$ and PM_{10}.

DATES: The conference call meeting will take place on Thursday, May 1, 2003, from 10 a.m. to 12 p.m. eastern time. Participation will be by teleconference only.

ADDRESSES: Members of the public who wish to obtain the call-in number and access code to participate must contact Ms. Delores Darden, EPA Science Advisory Board Staff, at telephone/voice mail: (202) 564–2282, via e-mail at: *darden.delores@epa.gov;* or at mailing address: EPA Science Advisory Board, U.S. Environmental Protection Agency (1400A), 1200 Pennsylvania Avenue, NW., Washington, DC, 20460 (FedEx/Courier Zip Code: 20004), in order to register.

FOR FURTHER INFORMATION CONTACT: Any member of the public wishing further information about this conference call should contact Mr. Fred Butterfield, Designated Federal Officer (DFO), EPA Science Advisory Board Staff; at telephone/voice mail: (202) 564–4561; or via e-mail at: *butterfield.fred@epa.gov.* General information concerning the CASAC or the EPA Science Advisory Board can be found on the EPA Web site at: *http://www.epa.gov/sab.*

SUPPLEMENTARY INFORMATION:

1. Summary. The Clean Air Scientific Advisory Committee was established by 42 U.S.C. 7409 in part to provide advice, information and recommendations on the scientific and technical aspects of issues related to the criteria for national ambient air quality standards (NAAQS). The CASAC Particulate Matter Review Panel will report to the Administrator of EPA through the CASAC, which is

administratively located under the EPA Science Advisory Board. The SAB was established by 42 U.S.C. 4365 to provide independent scientific and technical advice, consultation, and recommendations to the EPA Administrator on the technical basis for Agency positions and regulations. Both the CASAC and the SAB are Federal advisory committees chartered under the Federal Advisory Committee Act (FACA), as amended (5 U.S.C. App.). The CASAC Particulate Matter Review Panel will comply with the provisions of FACA and all appropriate SAB procedural policies.

On April 9, 2003, EPA's Office of Air Quality Planning and Standards (OAQPS) will make available for public review and comment a draft memorandum, ''Preliminary Recommended Methodology for PM_{10} and $PM_{10–2.5}$ Risk Analyses in Light of Reanalyzed Study Results'' (hereafter, draft Risk Analysis Methodology for PM_{10} and $PM_{10–2.5}$). This document outlines the overall scope proposed for the quantitative risk assessments for PM_{10} and coarse-fraction PM ($PM_{10–2.5}$) that will be conducted as part of the periodic review of the NAAQS for PM, pursuant to sections 108 and 109 of the Clean Air Act (CAA).

2. Background. On January 28, 2002 (67 FR 3897), OAQPS made available for public and CASAC review a draft document, ''Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas'' (hereafter, draft PM Risk Analysis Methodology), that describes EPA's plans and approach for conducting PM health risk analyses primarily for fine particles ($PM_{2.5}$). The PM risk analyses will be performed to assist in the preparation of the OAQPS PM Staff Paper, the purpose of which is to evaluate the policy implications of the key scientific and technical information contained in the Agency's PM Air Quality Criteria Document (AQCD) and identify critical elements that EPA staff believe should be considered in reviewing the PM NAAQS. The Staff Paper is intended to ''bridge the gap'' between the scientific review contained in the AQCD and the public health and welfare policy judgments required of the Administrator in reviewing the NAAQS. On February 27, 2002, the CASAC PM Review Panel met via public teleconference to provide advice to EPA on the proposed methodology; and, on May 23, 2002, the CASAC issued an Advisory providing its advice to the EPA Administrator entitled, ''Review of the Agency's Draft Proposed Methodology for Particulate Matter Risk Analysis for Selected Urban Areas; an Advisory by the Clean Air Scientific Advisory Committee (EPA–SAB–CASAC–ADV–02–002), located on the EPA Science Advisory Board Web site at: *http://www.epa.gov/sab/pdf/casacadv02002.pdf.*

In response to the advice provided in the May 2002 CASAC Advisory, OAQPS has proposed to expand the scope of the PM health risk analyses to include risk analyses for PM_{10}. The charge to the CASAC PM Panel during their consultation on May 1, 2003, is to provide feedback on the scope and approach proposed by EPA for the PM_{10} and $PM_{10–2.5}$ components of the risk analyses. EPA is making available the draft Risk Analysis Methodology for PM_{10} and $PM_{10–2.5}$ to facilitate discussion and review of the proposed approach by the CASAC and general public. This draft document takes into consideration the availability of reanalyses using alternative statistical approaches for some PM health effect studies identified by EPA as being of high priority for policy considerations (*see* the following URL: *http://www.epa.gov/ncea/partmatt.htm,* for more information). This document outlines the overall scope proposed for the quantitative risk assessments for PM_{10} and $PM_{10–2.5}$ including health endpoints to be analyzed, health studies that serve as the source of concentration-response functions, and cities to be examined.

Following the May 1, 2003, CASAC Particulate Matter Review Panel teleconference to review the draft Risk Analysis Methodology for PM_{10} and $PM_{10–2.5}$, EPA will prepare a technical report describing the risk analysis methodology in greater detail and including preliminary risk estimates taking into account public and CASAC comments. The methodology and preliminary estimates will be summarized in the next draft of the OAQPS PM Staff Paper, which will be released for public and CASAC review later this year.

Any questions concerning the draft Risk Analysis Methodology for PM_{10} and $PM_{10–2.5}$ should be directed to Mr. Harvey Richmond, OAQPS's Health and Ecosystems Effects Group, at telephone/voice mail: (919) 541–5271; or via e-mail at: *richmond.harvey@epa.gov.*

3. Availability of Additional Meeting Materials. A copy of the draft memorandum, ''Preliminary Recommended Methodology for PM_{10} and $PM_{10–2.5}$ Risk Analyses in Light of Reanalyzed Study Results'' will be available through EPA's Technology Transfer Network (TTN) Web site under the technical area for National Ambient Air Quality Standards, under the heading of ''Particulate Matter—Technical Documents'' at the following URL address: *http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_td.html* after April 9, 2003. In addition, the draft agenda for the teleconference that is the subject of this notice will be posted on the EPA Science Advisory Board Web Site at: *http://www.epa.gov/sab* (under the ''Agendas'' subheading) approximately 10 days before the publically-accessible teleconference.

4. Providing Oral or Written Comments at SAB Meetings. It is the policy of the EPA Science Advisory Board (SAB) to accept written public comments of any length, and to accommodate oral public comments whenever possible. The EPA SAB expects that public statements presented at its meetings will not be repetitive of previously submitted oral or written statements. *Oral Comments:* In general, each individual or group requesting an oral presentation at a face-to-face meeting will be limited to a total time of 10 minutes (unless otherwise indicated). For conference call meetings, opportunities for oral comment will usually be limited to no more than three minutes per speaker and no more than 15 minutes total. Interested parties should contact the CASAC DFO, Mr. Fred Butterfield, at the telephone number or e-mail address provided above, at least one week prior to the meeting in order to be placed on the public speaker list for the meeting. Speakers may attend the meeting and provide comment up to the meeting time. Speakers should bring at least 35 copies of their comments and presentation slides for distribution to the reviewers and public at the meeting. *Written Comments:* Although the SAB accepts written comments until the date of the meeting (unless otherwise stated), written comments should be received in the SAB Staff Office at least one week prior to the meeting date so that the comments may be made available to the review panel for their consideration. Written comments should be supplied to Ms. Delores Darden, EPA Science Advisory Board Staff, at the e-mail address or mailing address provided above, or via fax at: (202) 501–0582, in the following formats: one hard copy with original signature, and one electronic copy via e-mail (acceptable file format: Adobe Acrobat, WordPerfect, Word, or Rich Text files (in IBM–PC/Windows 95/98 format). Those providing written comments and who attend the meeting are also asked to bring 35 copies of their comments for public distribution. Any written comments supplied at the meeting should be provided to the DFO up to or

immediately following the meeting. The SAB allows a grace period of 48 hours after adjournment of the public meeting to provide written comments supporting any verbal comments stated at the public meeting to be made a part of the public record.

5. *Meeting Access.* Individuals requiring special accommodation to access this teleconference should contact Ms. Delores Darden, EPA Science Advisory Board Staff, at the telephone or e-mail address provided above, at least five business days prior to the meeting so that appropriate arrangements can be made.

Dated: April 7, 2003.

Vanessa T. Vu,

Director, EPA Science Advisory Board Staff Office.

[FR Doc. 03–9040 Filed 4–11–03; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–7483–1]

Notice of Extension of Public Comment Period on the Draft Final Guidelines for Carcinogen Risk Assessment and the Draft Supplemental Guidance for Assessing Cancer Susceptibility From Early-Life Exposure to Carcinogens

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of extension of public comment period.

SUMMARY: This notice extends the comment period for the Draft Final Guidelines for Carcinogen Risk Assessment and the draft Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens. The availability of these documents was originally announced in the **Federal Register** on March 3, 2003 (68 FR 10012).

DATES: Comments must be received by Monday, June 2, 2003.

ADDRESSES: The documents are available via the Internet from *www.epa.gov/ncea/raf/cancer2003.htm*. Instructions for submitting comments are provided at this website and in the March 3, 2003 **Federal Register** notice.

FOR FURTHER INFORMATION CONTACT: Dr. William P. Wood, Risk Assessment Forum (mail code 8601D), U.S. Environmental Protection Agency, 1200 Pennsylvania Avenue NW., Washington, DC 20460, telephone 202–564–3361, or send electronic mail inquiries to *risk.forum@epa.gov*.

SUPPLEMENTARY INFORMATION: In the March 3, 2003 **Federal Register** (68 FR 10012), EPA announced the availability of, and opportunity to comment on, the Draft Final Guidelines for Carcinogen Risk Assessment (February 2003, NCEA–F–0644A) and the draft Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens (EPA/630/R–03/003). The comment period was scheduled to close on May 1, 2003. This notice extends the comment period until June 2, 2003. EPA will consider all comments received by this date in completing final Guidelines and supplemental guidance.

As announced in the **Federal Register** on April 11, 2003, a panel of EPA's Science Advisory Board (SAB) will meet to review the draft Supplemental Guidance on May 12 to 14, 2003. EPA will provide all public comments on the draft Supplemental Guidance that EPA has received by May 1, 2003 to the SAB review panel prior to its meeting. Comments received by EPA by June 2, 2003 but after May 1, 2003 will also be forwarded to the SAB for consideration by the review panel in completing its report. Comments may also be submitted directly to the SAB in the manner described in the **Federal Register** notice announcing the SAB meeting. It is the policy of the SAB to accept written comments and accommodate oral public comments wherever possible at its public meetings.

Dated: April 8, 2003.

Paul Gilman,

Assistant Administrator for Research and Development.

[FR Doc. 03–9048 Filed 4–11–03; 8:45 am]

BILLING CODE 6560–50–P

FEDERAL COMMUNICATIONS COMMISION

Notice of Public Information Collection(s) Being Reviewed by the Federal Communications Commission

April 4, 2003.

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act of 1995, Public Law 104–13. An agency may not conduct or sponsor a collection of information unless it displays a current valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the Paperwork Reduction Act (PRA) that does not display a valid control number. Comments are requested concerning (a) whether the proposed collection of information is necessary for the proper performance of the functions of the Commission, including whether the information shall have practical utility; (b) the accuracy of the Commission's burden estimate; (c) ways to enhance the quality, utility, and clarity of the information collected; and (d) ways to minimize the burden of the collection of information on the respondents, including the use of automated collection techniques or other forms of information technology.

DATES: Written comments should be submitted on or before June 13, 2003. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of time allowed by this notice, you should advise the contact listed below as soon as possible.

ADDRESSES: Direct all comments to Les Smith, Federal Communications Commission, Room 1–A804, 445 12th Street, SW., Washington, DC 20554, or via the Internet to *Leslie.Smith@fcc.gov.*

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collection(s) contact Les Smith at 202–418–0217 or via the Internet at *Leslie.Smith@fcc.gov.*

SUPPLEMENTARY INFORMATION:

OMB Control Number: 3060–0208.

Title: Section 73.1870, Chief Operators.

Form Number: N/A.

Type of Review: Extension of a currently approved collection.

Respondents: Business and other for-profit entities; Not-for-profit institutions.

Number of Respondents: 14,500.

Estimated Time per Response: 26 hours.

Frequency of Response: Recordkeeping; Third party disclosure.

Total Annual Burden: 379,407.

Total Annual Costs: $0.00.

Needs and Uses: 47 CFR 73.1870 requires that the licensee of an AM, FM, or TV broadcast station designate a chief operator of the station. Section 73.1870(b)(3) requires that this designation must be in writing and posted with the station license. Section 73.1230 requires that all licensees post station licenses ''at the place the licensee considers the principal control point of the transmitter'' generally at the transmitter site. Agreements with chief operators serving on a contract basis must be in writing with a copy kept in

Attachment D

Proposed Plans for Particulate Matter Health Risk Analyses for Coarse Particulate Matter ($PM_{10-2.5}$) and PM_{10}

Harvey Richmond, U.S. EPA

Ellen Post, Leland Deck, Abt Associates, Inc.

May 1, 2003 Presentation to CASAC

Background

- As Part of Last PM NAAQS Review (1996/1997) EPA Conducted PM Risk Analyses for 2 Urban Counties (Philadelphia & LA)
- CASAC Consultation on Draft Scoping Plan – July 2001
- CASAC Teleconference on $PM_{2.5}$ Risk Methodology – February 27, 2002 and May 23, 2002 Advisory
  - General methodology appropriate, various comments on details of application
  - Encouraged EPA to conduct PM_{10} risk analyses for wider range of health effects in cities with relatively greater and lesser effects to "provide a valuable perspective for the likely upper bound of the health impact of $PM_{2.5}$ and help provide some measure of the variability of risk across a wider range of conditions than the eight cities afford."
  - Agreed should hold a consultation meeting on plans for $PM_{10-2.5}$ risk analyses

Proposed Plans for $PM_{10-2.5}$ and PM_{10} Health Risk Analyses

- General methodology is same as described in prior $PM_{2.5}$ risk analysis methodology report (January 2002, Abt Assoc.)
  - For $PM_{10-2.5}$ initially plan on analyzing recent year of air quality (alternative standards to be analyzed later)
  - For PM_{10} plan on only analyzing recent year of air quality
- Scope of health risk analyses for $PM_{10-2.5}$ and PM_{10}
  - Selection of health endpoint categories
  - Selection of urban areas to examine
  - Selection of epidemiology studies
  - Selection of concentration-response relationships to include in analyses

Criteria and Selection of Health Endpoints

- Focus on more severe and better understood endpoints
- Where weight of the evidence (based on PM CD) supports likely causal relationship

- PM_{10}
  - Mortality (total & cause specific)
  - Hospital admissions (and possibly ER visits)
  - Respiratory symptoms
- $PM_{10-2.5}$
  - Hospital admissions (and possibly ER visits)
  - Respiratory symptoms

Criteria and Selection of Urban Areas

-Sufficient air quality data for recent year (1999 or later)

-Same urban area or close to locations where epidemiological studies conducted

-For hospital admissions, need recent year of baseline incidence data

-For PM_{10} prefer to include (1) areas that inform comparisons both across PM indicators and across health effects, (2) areas across various parts of U.S., and (3) areas where studies have relatively greater statistical power

- PM_{10}
  - Boston, MA
  - Chicago, IL
  - Detroit, MI
  - Los Angeles, CA
  - Minneapolis-St. Paul, MN
  - Philadelphia, PA
  - Phoenix, AZ
  - Provo, UT
  - San Jose, CA
  - Seattle, WA
  - St. Louis, MO

- $PM_{10-2.5}$
  - Detroit
  - St. Louis

- $PM_{2.5}$ (for comparison)
  - Boston
  - Detroit
  - Los Angeles
  - Philadelphia
  - Phoenix
  - San Jose
  - Seattle
  - St. Louis

Criteria for Selection of Studies

- Acceptable, published, peer-reviewed studies evaluated in draft PM CD
- Study directly measured PM using PM_{10} or $PM_{10-2.5}$ as indicator
- C-R relationships from studies not involving GAM/S-Plus issue and from peer-reviewed reanalyses of GAM studies

Table 1. Currently Available Health Endpoints and Urban Locations for Short-Term Exposure

Urban Locations	S.T. Total Mortality	S.T. Cardioresp Mort.	S.T. Cardiovasc Mort.	S.T. Circ. Mort.	S.T. Resp Mort.	Hosp Adm Total Resp.	Hosp. Adm. COPD	Hosp. Adm. Pneum	Hosp. Adm. Asthma	Hosp. Adm. Cardio.	ER Visits **	Resp Sympt.
Boston	F, T											F, T
Chicago	T	T	T	T	T		T	T		T		
Detroit	F, T	T		F, T	F, T		F, T,C	F, T,C		F, T,C		
Los Angeles	F, T	T	F, T		F, T	T	F, T		T	F, T		T***
Minneapolis-St. Paul	T	T					T	T		T		
Philadelphia	F, T	T	F		F							
Phoenix	T	T	F, T									
Provo	T		T		T		T	T		T		T
San Jose	F, T	T	F, T		F, T						T	
Seattle	T	T					T	T	T	T	T***	T***
St.Louis	F, T											F, T,C

*F = $PM_{2.5}$, T = PM_{10}, C = $PM_{10-2.5}$
**Need emergency room visits baseline incidence
***Added since April 8, 2003 Draft Abt Memo

Proposed Approach for Selecting C-R Relationships

- Include both single and multi-pollutant functions where available
- Include both single and multi-city functions where available (e.g., for PM_{10} mortality C-R from NMMAPS, use single city and regional C-R functions)
- For reanalyzed "GAM" studies, use GAM C-R function with more stringent convergence criteria (per EPA guidance)
- Lags
  - Use lag(s) recommended by authors in study or the draft PM CD
  - Include distributed lag if feasible
- For two example cities (Chicago and Los Angeles), include multiple models (various GAM, GLM) and lags (0 to 5 days)

Issues for Consultation

- Panel members views on the criteria and rationale presented for selection of proposed health endpoints, urban locations, and health studies to be included in the PM_{10} and $PM_{10-2.5}$ risk analyses

- Panel members views on the range of concentration-response relationships to be included in the two example urban locations

Attachment E

memorandum

Environmental Research Area
4800 Montgomery Lane, Suite 600 # Bethesda, MD 20814-5341 # (301) 913-0500

Abt Associates Inc.

Date April 8, 2003 (Draft)

To Harvey Richmond, U.S. EPA/OAQPS

From Ellen Post, Abt Associates Inc.

Subject **Preliminary Recommended Methodology for PM_{10} and $PM_{10-2.5}$ Risk Analyses in Light of Reanalyzed Study Results**

The basic methodology for the proposed PM_{10} and $PM_{10-2.5}$ health risk analyses is very similar to the methodology used for the $PM_{2.5}$ risk analyses, described in detail in the Abt Associates draft technical support document (TSD), "Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas," dated January, 2002. The discussion of methodology in that draft TSD included

- an overview of the methods that we propose to use and the assumptions upon which the analyses are based, covering (1) the basic structure of the risk analyses, (2) air quality inputs, (3) simulating just meeting PM standards, (4) baseline incidence data, (5) calculation of health effects incidence, (6) characterization of uncertainties, and (7) proposed sensitivity analyses;

- a discussion of the health endpoints included, and the rationale for including them, as well as a discussion of the locations selected for the risk analyses and the rationale for choosing these locations;

- a discussion of how we selected one (or more) concentration-response (C-R) function for those health endpoints for which more than one C-R function is available;

- a discussion of baseline health effect incidence rates; and

- a discussion of sources of uncertainty.

There are a few aspects of the methodology for the proposed PM_{10} and $PM_{10-2.5}$ health risk analyses, however, that require further elaboration. First, the health endpoints, locations and studies included, and the rationale for including them, are specific to the PM_{10} and $PM_{10-2.5}$ health risk analyses. Second, the required air quality inputs will also be specific to the proposed PM_{10} and $PM_{10-2.5}$ health risk analyses. Each of these is discussed in turn below.

In addition, in response to comments made by the CASAC in February 2002 on the proposed methodology described in the January 2002 draft TSD, we propose to add two sensitivity analyses to those proposed for the $PM_{2.5}$ health risk analyses in that draft TSD. (We will also add these two sensitivity analyses to the revised $PM_{2.5}$ health risk analyses, to be described in a revised draft TSD.) These sensitivity analyses will address variability in background concentrations and seasonal concentration-response (C-R) relationships. They are described more fully below.

1. Selection of Health Endpoints, Urban Areas, and Studies

OAQPS staff carefully reviewed the evidence evaluated in the Third External Review Draft of Air Quality Criteria for Particulate Matter (April, 2002) (hereafter, 2002 draft PM CD). Tables 8A-1 and 8A-2 in the 2002 draft PM CD summarize the available U.S. and Canadian short-term exposure studies that provide effect estimates for all PM indicators for mortality and morbidity, respectively. Table 9-15 in the 2002 draft PM CD summarizes the available U.S. and Canadian short-term exposure studies specifically on $PM_{10-2.5}$. We are not proposing to conduct any PM_{10} or $PM_{10-2.5}$ risk analyses based on long-term exposure studies. The weight of the evidence presented in the draft PM CD suggests that the component of PM_{10} that is most likely associated with long-term exposure mortality is the fine fraction, $PM_{2.5}$. (We are including both short-term and long-term exposure studies in the $PM_{2.5}$ risk analyses.)

Health effect categories

We propose to include in the quantitative PM_{10} and $PM_{10-2.5}$ risk analyses only the more severe and better understood (in terms of health consequences) health endpoint categories for which the weight of the evidence supports the existence of a likely causal relationship between various PM indicators and the effect category. For these health effect categories, the risk analyses will be predicated on the assumption that the relationships are causal. In addition, only those categories which include studies that satisfy the study selection criteria (see below) will be included.

Urban areas

An urban area can be included in the proposed PM_{10} or $PM_{10-2.5}$ risk analyses only if it satisfies the following criteria:

- It has sufficient air quality data for a recent year (1999 or later) A city will be considered to have sufficient PM_{10} air quality data if it had at least one PM_{10} monitor at which there were at least 11 observations per quarter for a one year period. Sufficient air quality data for $PM_{10-2.5}$ is defined as a one year period with at least 11 daily values per quarter based on data from co-located PM_{10} and $PM_{2.5}$ monitors.[1]

- It is in the United States.

- It is the same as or close to the location where at least one C-R function for one of the recommended health endpoints (see below) has been estimated by a study that satisfies the study selection criteria (see below).[2]

- For the hospital admission effects category, the availability of relatively recent baseline incidence data, specific to International Classification of Disease (ICD) codes is necessary.[3]

Studies

Many studies, especially those carried out in recent years, fitted generalized additive models (GAM) to their time-series data. In late May 2002, EPA was informed by the Health Effects Institute (HEI) of a generally unappreciated aspect in the use of S-Plus statistical software often employed to fit these models. Using appropriate modifications of the default convergence criteria code in the S-Plus

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[1]To be consistent with the epidemiological studies which generally focus on using only population-oriented monitors, we will exclude from consideration any monitors where the monitoring objective was listed as "highest concentration monitor." The few monitors that would thus be excluded are sited in industrial or commercial areas and are intended to characterize local conditions near major point sources.

[2] Urban locations for which C-R functions were estimated often include several counties. (For example, in Klemm et al., 2000, the urban area labeled "Boston" consists of three counties: Middlesex, Norfolk, and Suffolk counties.) To the extent possible, in the PM risk analyses we will try to include the specific counties used in the urban location in the original epidemiological studies.

[3] The absence of hospital admissions baseline incidence data does not necessarily mean that we cannot use an urban area in the risk analysis, only that we cannot use it for the hospital admissions endpoint. Because comparisons across health effect categories is an additional consideration in the selection of urban areas for the PM_{10} risk analyses, however, an urban area could be excluded because of the lack of baseline incidence data.

software and a correct approach to estimating the variance of estimators will change the estimated C-R functions and could change the results of tests of significance of estimates, although it is not possible to predict *a priori* how estimates and significance tests will change. Many but not all of the C-R functions that were originally estimated using the S-Plus software for fitting GAMs have since been re-estimated using revised methods.

A study that has estimated one or more C-R functions for a recommended health endpoint in an urban location proposed to be used for the PM_{10} and/or $PM_{10-2.5}$ risk analyses must satisfy the following criteria:

- It is an acceptable, published, peer-reviewed study that has been evaluated by the 2002 draft PM CD.

- It directly measured PM using PM_{10} or $PM_{10-2.5}$ as the indicator.

- It either did not rely on GAMs using the S-Plus software to estimate C-R functions or has appropriately re-estimated them using revised methods.

In addition to the criteria discussed above, some additional considerations, specific to either the PM_{10} or the $PM_{10-2.5}$ risk analyses, were taken into account in the selection of urban areas. These, along with the resulting selection of health effects categories, urban areas, and studies, are detailed below, separately for the PM_{10} and $PM_{10-2.5}$ risk analyses in sections 1.1 and 1.2, respectively.

1.1 Health endpoints, urban areas, and studies proposed for the PM_{10} risk analyses

Based on OAQPS's review of the evidence evaluated in the 2002 draft PM CD, we propose to include the following broad categories of health endpoints associated with short-term exposures in the PM_{10} risk analyses:

- mortality (total and cause-specific)
- hospital admissions (and possibly emergency room visits) for cardiovascular and respiratory causes
- respiratory symptoms not requiring hospitalization.

Other effects reported to be associated with PM_{10} identified in the draft 2002 PM CD, such as decreased lung function, will be addressed qualitatively in the OAQPS PM Staff Paper.

In addition to the criteria listed above, the selection of urban areas that we propose to include in the PM_{10} risk analysis is further guided by the following considerations:

- Among its comments on the $PM_{2.5}$ risk analyses, the CASAC recommended that EPA expand its PM risk analyses for the current review to include PM_{10} risk analyses and to select cities across various parts of the United States.

- In addition, we would also like to include urban areas that would further inform comparisons both across the PM indicators (i.e.., $PM_{2.5}$, PM_{10}) and across health effects (e.g., mortality, hospital admissions).

- In light of these recommendations, we propose to include, at a minimum, those urban areas already selected for the $PM_{2.5}$ risk analyses (i.e., Boston, Detroit, Los Angeles, Philadelphia, Phoenix, San Jose, Seattle, and St. Louis), for which city-specific C-R functions for short-term exposure mortality are available from the NMMAPS study and/or other studies.

- Further, in selecting any additional urban areas, areas for which there are C-R functions with greater statistical power are preferred

Among studies that estimated C-R functions in locations for which there is sufficient air quality data, the statistical power of a study is an important consideration. In general, the power of a study increases as the number of its observations increases. The number of observations depends not only on the number of days on which health effect counts were obtained, but also on the size of the counts. The 2002 draft PM CD uses the natural logarithm of the mortality-days (i.e., the natural log of the product of the number of study days and the average number of deaths per day) as a surrogate or indicator reflecting the power of short-term exposure mortality epidemiological studies. In considering additional urban areas, we will consider only those urban areas in which studies with relatively greater statistical power were conducted. Specifically, for C-R functions for mortality from short-term exposure, we propose to consider only those studies that have a natural log of mortality-days greater than or equal to 9.0. This is the same statistical power criterion that we used in the $PM_{2.5}$ risk analyses.[4]

Based on the above criteria and considerations, we currently propose to include the following urban areas in the PM_{10} risk analyses:

- Boston, MA
- Chicago, IL
- Detroit, MI

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[4]Most of the epidemiological studies reporting total non-accidental mortality, also report on one or more cause specific mortality categories; in such studies the natural log of mortality days is often less than 9.0 because there are fewer deaths from a specific cause. Following the method used in the $PM_{2.5}$ risk analyses, we propose to include the cause-specific mortality C-R relationships reported in such studies as long as the natural log of total mortality days was greater than or equal to 9.0.

- Los Angeles, CA
- Minneapolis-St. Paul, MN
- Philadelphia, PA
- Phoenix, AZ
- Provo, UT
- San Jose, CA
- Seattle, WA
- St. Louis, MO

Most of these urban areas allow comparison both across PM indicators ($PM_{2.5}$ and PM_{10}) and across different health endpoints. While Chicago, Minneapolis-St. Paul, and Provo do not provide comparisons between $PM_{2.5}$ and PM_{10}, they do provide comparisons across health endpoints.

Exhibits 1 and 2 show the studies that are potentially available to be used in the PM_{10} health risk analyses for each of these urban areas for mortality and morbidity endpoints, respectively. Some of these studies will become available, however, only after they have been reanalyzed to address the S-Plus issue. Studies are classified into three groups:

- Studies that did not use GAM/S-Plus are shown in regular type; these studies can be included in the risk analyses.
- Studies that used GAM/S-Plus but were reanalyzed using revised methods are shown in bold; these studies are also currently available to be included.
- Studies that used GAM/S-Plus but have not yet been reanalyzed are shown in italics. These studies are *potentially* available – they will become available if they are reanalyzed.

We are not currently proposing to include Atlanta (shown in Exhibit 1) or Pittsburgh (shown in Exhibits 1 and 2), since PM_{10} studies that would provide the basis for comparisons across health endpoints have not been reanalyzed and, thus are not currently available for use in the PM_{10} risk analyses.

Many studies shown in Exhibits 1 and 2 estimated more than one C-R function (e.g., one single pollutant model and one or more multi-pollutant models). Several researchers reanalyzed some but not all of the C-R functions that they had originally estimated using the S-Plus software. It was typical, for instance, to reanalyze single pollutant models but not yet multi-pollutant models. A study that reanalyzed at least one C-R function for a health endpoint is shown in bold, even if other C-R functions for that health endpoint have not yet been reanalyzed.

Where both single and multi-pollutant models are available for a health endpoint in a given location, we propose to use both, as we similarly proposed for the $PM_{2.5}$ risk analyses. In some cases, however, this will not be possible. For those studies that used GAM/S-Plus and have reanalyzed only some of the C-R functions originally estimated (e.g., only the single pollutant functions), only those

models that have been reanalyzed will be included, as noted above. Where a C-R function has been estimated in a single city and a multi-city C-R function has been estimated which includes that city, both the single-city and the multi-city C-R functions will be included.[5]

[5] Regional results for mortality have been reanalyzed in the NMMAPS study, along with city-specific results. In addition, Schwartz (2003) has reanalyzed mortality results in 10 cities jointly. These multi-city functions can be applied in those cities included in the functions.

Exhibit 1. Mortality Endpoints, Urban Locations, and Studies Potentially Available for Use in the PM_{10} Risk Analyses

Urban Location	Short-Term Exposure Mortality Endpoint				
	Total (non-accidental)	**Cardiorespiratory**	**Cardiovascular**	**Circulatory**	**Respiratory/COPD**
Atlanta, GA	**Samet et al. (2000)***	*Samet et al. (2000)*			
Boston, MA	**Klemm et al. (2000)**				
Chicago, IL	Ito and Thurston (1996) **Moolgavkar (2000a)** **Samet et al. (2000)*** *Schwartz (2001)* Schwartz (2003)** Styer et al. (1995)	*Samet et al. (2000)*	**Moolgavkar (2000a)**	Ito and Thurston (1996)	Ito and Thurston (1996) **Moolgavkar (2000a)**
Detroit, MI	**Lippmann et al. (2000)** **Samet et al. (2000)*** Schwartz (2003)**	*Samet et al. (2000)*		**Lippmann et al. (2000)**	**Lippmann et al. (2000)**
Los Angeles, CA	Kinney et al. (1995) **Moolgavkar (2000a)** **Samet et al. (2000)***	*Samet et al. (2000)*	**Moolgavkar (2000a)**		**Moolgavkar (2000a)**
Minneapolis-St. Paul, MN	**Samet et al. (2000)*** Schwartz (2003)**	*Samet et al. (2000)*			
Philadelphia, PA	Lipfert et al. (2000)*** **Samet et al. (2000)***	*Samet et al. (2000)*			
Phoenix, AZ	*Mar et al. (2000)* **Samet et al. (2000)***	*Samet et al. (2000)*	**Mar et al. (2000)** *Moolgavkar (2000a)*		*Moolgavkar (2000a)*
Pittsburgh, PA	**Samet et al. (2000)***	*Samet et al. (2000)*			
Provo, UT	Pope et al. (1999)		Pope et al. (1999)		Pope et al. (1999)

Urban Location	Short-Term Exposure Mortality Endpoint				
	Total (non-accidental)	**Cardiorespiratory**	**Cardiovascular**	**Circulatory**	**Respiratory/COPD**
San Jose, CA	**Fairley (1999)** **Samet et al. (2000)***	*Samet et al. (2000)*	**Fairley (1999)**		**Fairley (1999)**
Seattle, WA	**Samet et al. (2000)*** Schwartz (2003)**	*Samet et al. (2000)*			
St. Louis, MO	**Klemm et al. (2000)**				

Note: Regular type indicates that the study did not use GAM/S-Plus; bold indicates that it used GAM/S-Plus but has been reanalyzed using revised methods; and italics indicates that the study used GAM/S-Plus and has not yet been reanalyzed.

*Reanalysis results were obtained from the HEI website at http://www.biostat.jhsph.edu/biostat/research/web.est.xls on March 13, 2003.

**Schwartz (2003) is a reanalysis of results in three earlier studies, to address the GAM/S-Plus issue. As part of this reanalysis, Schwartz estimated a single multi-city C-R function for short-term exposure mortality and daily deaths in 10 U.S. cities (see Table 2 in the paper), including four of the cities (Chicago, Detroit, Minneapolis, and Seattle) that we propose to include in our PM_{10} risk analyses.

***We currently do not have upper and lower bounds on the coefficient in the Lipfert study. We requested these from the authors and are currently uncertain as to whether this information will be provided in time for the PM risk analyses. We cannot use this study unless we obtain these upper and lower bounds.

Exhibit 2. Morbidity Endpoints, Urban Locations, and Studies Potentially Available for Use in the PM_{10} Risk Analyses

Urban Location	Hospital Admissions (total respiratory)	Hospital Admissions (COPD)	Hospital Admissions (Pneumonia)	Hospital Admissions (Asthma)	Hospital Admissions (Cardiovascular)	Emergency Room Visits (Asthma)	Respiratory Symptoms
Boston, MA							Schwartz et al. (1994)
Chicago, IL		**Moolgavkar (2000c)** *Samet et al. (2000)*	*Samet et al. (2000)*		**Moolgavkar (2000b)** Morris and Naumova (1998) *Samet et al. (2000)* *Schwartz (1999)*		
Detroit, MI		**Lippmann et al. (2000)** *Samet et al. (2000)* Schwartz (1994a)	**Lippmann et al. (2000)** *Samet et al. (2000)* Schwartz (1994a)		**Lippmann et al. (2000)*** *Samet et al. (2000)* Schwartz and Morris (1995)*		
Los Angeles, CA	Linn et al. (2000)	Linn et al. (2000) **Moolgavkar (2000c)**		Linn et al. (2000) Nauenberg and Basu (1999)**	Linn et al. (2000) **Moolgavkar (2000b)**		
Minneapolis-St. Paul		*Moolgavkar et al. (1997)* *Samet et al. (2000)* Schwartz (1994c)	*Moolgavkar et al. (1997)* *Samet et al. (2000)* Schwartz (1994c)		*Samet et al. (2000)* *Schwartz (1999)*		
Phoenix, AZ		*Moolgavkar (2000c)*			*Moolgavkar (2000b)*		
Pittsburgh, PA		*Samet et al. (2000)*	*Samet et al. (2000)*		*Samet et al. (2000)*		
Provo, UT		*Samet et al. (2000)*	*Samet et al. (2000)*		*Samet et al. (2000)*		Pope et al. (1991)

Urban Location	Hospital Admissions (total respiratory)	Hospital Admissions (COPD)	Hospital Admissions (Pneumonia)	Hospital Admissions (Asthma)	Hospital Admissions (Cardiovascular)	Emergency Room Visits (Asthma)	Respiratory Symptoms
San Jose, CA						Lipsett et al. (1997)***	
Seattle, WA		*Moolgavkar et al. (2000)* *Samet et al. (2000)*	*Samet et al. (2000)*	**Sheppard et al. (1999)**	*Samet et al. (2000)* *Schwartz (1999)*		
St. Louis, MO							Schwartz et al. (1994)

Note: Regular type indicates that the study did not use GAM/S-Plus; bold indicates that it used GAM/S-Plus but has been reanalyzed using revised methods; and italics indicates that the study used GAM/S-Plus and has not yet been reanalyzed.

*Lippmann et al. (2000) estimated separate C-R functions for hospital admissions for the following illnesses within the broad category of cardiovascular illnesses: Ischemic heart disease (ICD codes 410-414), dysrhythmias (ICD code 427), and congestive heart failure (ICD code 428). Schwartz and Morris (1995) estimated separate C-R functions for hospital admissions for ischemic heart disease (ICD codes 410-414) and congestive heart failure (ICD code 428).

**This study includes only emergency-related hospital admissions for asthma, excluding all scheduled admissions and transfers from other facilities. The model estimated is only for the wet season, from November 15 - March 1 (based on four years: 1991 - 1994).

***This study estimated a C-R function for ER visits for asthma. It presents results from a model including not only PM but also the interaction between PM and minimum temperature. We can use this study only if we obtain (1) daily minimum temperatures and (2) baseline incidence data for asthma ER visits.

1.2 Health endpoints, urban areas, and studies proposed for the $PM_{10-2.5}$ risk analyses

A number of studies have estimated C-R relationships between $PM_{10-2.5}$ and both non-accidental total mortality and cause-specific mortality (due to short-term exposure), and some of the more recent studies have reported positive and statistically significant results. However, based on the evaluation provided in the 2002 draft PM CD, OAQPS has judged that the weight of the evidence to date is not sufficient to support including short-term exposure mortality among the health endpoints in the $PM_{10-2.5}$ risk analyses. We therefore propose to include in the $PM_{10-2.5}$ risk analyses only the morbidity-related categories of health endpoints associated with short-term exposure proposed to be used in the PM_{10} risk analyses. This includes

- hospital admissions for cardiovascular and respiratory causes, and
- respiratory symptoms not requiring hospitalization.

Other morbidity effects reported to be associated with $PM_{10-2.5}$, identified in the draft 2002 PM CD, such as decreased lung function, will be addressed qualitatively in the OAQPS PM Staff Paper.

We would prefer to include urban areas in the $PM_{10-2.5}$ risk analyses for which we also plan to conduct $PM_{2.5}$ risk analyses, if there are epidemiological studies reporting associations for $PM_{10-2.5}$ in these locations. Because the $PM_{10-2.5}$ risk analyses require air quality data for PM_{10} and $PM_{2.5}$ at co-located monitors, the criterion of sufficient air quality data is significantly more limiting in the selection of urban areas for the $PM_{10-2.5}$ risk analyses than for either the PM_{10} or the $PM_{2.5}$ risk analyses.[6]

Based on these considerations, we currently propose to conduct $PM_{10-2.5}$ risk analyses for Detroit and St. Louis. While sufficient air quality data also are available for Los Angeles, the relevant epidemiological study has not been reanalyzed.

Exhibit 3 shows the studies potentially available to be used in the $PM_{10-2.5}$ health risk analyses for all three of these urban areas for morbidity endpoints. Studies are classified into the same three groups as before: (1) those that did not use GAM/S-Plus (shown in regular type); (2) those that used GAM/S-Plus but were reanalyzed using revised methods (shown in bold); and (3) those that used GAM/S-Plus but have not yet been reanalyzed (shown in italics).

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[6] We recently received year 2001 air quality data. The assessment of which locations have met the completeness criterion "for a recent year" is therefore based on air quality data in years 1999 through 2001. Although Boston was considered as a possible urban location for the $PM_{10-2.5}$ risk analyses, there were no co-located PM_{10} and $PM_{2.5}$ monitors in Boston that met the selection criterion in any of those years.

Exhibit 3. Health Endpoints, Urban Locations, and Studies Potentially Available for Use in the $PM_{10-2.5}$ Risk Analyses

Urban Location	Health Endpoint		
	Respiratory Hospital Admissions	**Ischemic Heart Disease Hospital Admissions**	**Respiratory Symptoms**
Detroit, MI	**Lippmann et al. (2000)**	**Lippmann et al. (2000)**	
Los Angeles, CA	*Moolgavkar (2000c)*		
St. Louis, MO			Schwartz and Neas (2000)*

Note: Regular type indicates that the study did not use GAM/S-Plus; bold indicates that it used GAM/S-Plus but has been reanalyzed using revised methods; and italics indicates that the study used GAM/S-Plus and has not yet been reanalyzed.
*A single C-R function was estimated in Schwartz and Neas (2000) based on combined data from six urban locations.

2. Air Quality Inputs

2.1. Estimating PM background levels

Since health risks will be calculated only for concentrations exceeding estimated background levels, estimates of background PM concentrations in the assessment locations are needed to calculate risk at "as is" concentrations in excess of background and for just meeting specified standards (for $PM_{10-2.5}$) attributable to concentrations exceeding background levels.

Consistent with the prior PM CD, the 2002 draft PM CD estimates background annual average PM_{10} concentrations to be in the range of 4 to 8 $\mu g/m^3$ in the Western United States and 5 to 11 $\mu g/m^3$ in the Eastern United States. We propose to use the midpoints of these ranges for the base case PM_{10} analysis. Thus background PM_{10} concentrations in the base case analysis will be estimated to be 8 $\mu g/m^3$ in the urban areas in the East (i.e., Boston, Philadelphia, Detroit, St. Louis, Atlanta, Chicago, and Minneapolis-St. Paul); and 6.0 $\mu g/m^3$ in those urban areas in the West (i.e., Los Angeles, San Jose, Phoenix, Seattle, and Provo).

The 2002 draft PM CD estimates background $PM_{2.5}$ concentrations to be in the range of 1 to 4 $\mu g/m^3$ in the Western United States and 2 to 5 $\mu g/m^3$ in the Eastern United States. Background $PM_{10-2.5}$ will be taken to be in the range of 3 (=4-1) to 4 (=8-4) $\mu g/m^3$ in the Western United States and 3 (=5-2) to 6 (=11-5) $\mu g/m^3$ in the Eastern United States.[7] We will use the midpoints of these ranges

[7] These ranges assume that the lowest background levels of PM_{10} and $PM_{2.5}$ occur in the same places, and similarly, the highest background levels of PM_{10} and $PM_{2.5}$ occur in the same places. While this assumption may not be true, we have no additional information on which to base ranges of background $PM_{10-2.5}$.

(3.5 :g/m^3 in the Western United States and 4.5 :g/m^3 in the Eastern United States) for the $PM_{10-2.5}$ base case analysis. Currently, however, we have only an Eastern city, Detroit, in the $PM_{10-2.5}$ risk analysis. Background $PM_{10-2.5}$ concentration in the base case analysis will be estimated to be 4.5 :g/m^3 in Detroit.

3. Sensitivity Analyses

In response to comments from the CASAC in February 2002, we propose to conduct two sensitivity analyses that were not included among those originally proposed for the $PM_{2.5}$ risk analyses. First, we propose to explore the impact of the assumption of a constant daily background level via sensitivity analyses. To assess the impact of using different daily background PM_{10} concentrations on the estimates of risk associated with "as is" PM_{10} concentrations in excess of background, two distributions of background levels, one for the East, and one for the West, will be developed. Each distribution will be lognormal with a mean equal to the midpoint of the range for that region of the country (see above) and a standard deviation based on the standard deviations of daily PM_{10} measurements from the Interagency Monitoring of Protected Visual Environments (IMPROVE) program. IMPROVE is a cooperative visibility monitoring effort between the EPA, federal land management agencies, and state air agencies. One of the functions of this program is to monitor visibility and aerosol conditions in Class I areas, and for the most part the IMPROVE monitors are located in rural areas. IMPROVE data from 1988 to 1999 will used in this analysis. An analogous procedure will be used for $PM_{10-2.5}$.

Second, we propose to explore the impact of estimating risk reductions on a seasonal rather than an annual basis, using seasonal PM_{10} C-R functions for mortality in San Jose estimated by Fairley (1999).[8] This will be carried out only for San Jose because that is the only location for which seasonal C-R functions were available that met the critiera for selection of health studies, endpoints, and urban locations. There were no studies providing C-R relationships on a seasonal basis for $PM_{10-2.5}$.

[8] Fairley (1999) used the S-Plus software to fit generalized additive models to time series data in San Jose. However, he reanalyzed not only the annual models but the seasonal models as well.

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Attachment F

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April 24, 2003

FILE NO: 31531.090001

Via Overnight Delivery and Electronic Mail

Ms. Delores Darden
EPA Science Advisory Board Staff
EPA Science Advisory Board
U. S. Environmental Protection Agency (1400A)
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20004

Re: Comments on EPA's Draft Preliminary Recommended Methodology for PM_{10} and $PM_{10-2.5}$ Risk Analyses in Light of Reanalyzed Study Results

Dear Ms. Darden:

Enclosed are the comments of Anne E. Smith, Ph.D., Vice President, Charles River Associates, on EPA's Draft Preliminary Recommended Methodology for PM_{10} and $PM_{10-2.5}$ Risk Analyses in Light of Reanalyzed Study Results. Dr. Smith prepared these comments on behalf of the Utility Air Regulatory Group.

These comments are being filed one week prior to the scheduled May 1, 2003 teleconference of the Clean Air Scientific Advisory Committee (CASAC). 68 Fed. Reg. 17939 (April 14, 2003). Please distribute these comments to the CASAC members for their consideration prior to the teleconference.

If you have any questions regarding the comments, please contact one of us.

Sincerely,

Allison D. Wood

Lucinda Minton Langworthy
Allison D. Wood

Counsel for the Utility Air Regulatory Group

COMMENTS ON EPA'S "PROPOSED METHODOLOGY FOR PARTICULATE MATTER RISK ANALYSES FOR SELECTED URBAN AREAS"

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Prepared on behalf of the Utility Air Regulatory Group
February 27, 2002

The U.S. Environmental Protection Agency (EPA) released its *Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas* in January 2002 and has requested public comment. This document provides comments on behalf of the Utility Air Regulatory Group (UARG) on this risk analysis plan (the "Plan").

This draft Risk Analysis Plan has given balance to some of its discussions of the options for quantitatively estimating risk using the body of epidemiological literature. One example is that the draft now recognizes the merits of multi-pollutant models, and no longer plans to use only single-pollutant models. However, it is important that balance find its way into the actual analysis and also that the analysis be designed in a way that will lead to balance in communication of the final results. My comments are focused on these concerns.

My central comment is that the planned risk analysis will be focused on a narrow subset of information and assumptions that are the most consistent with the regulatory judgments EPA has made on $PM_{2.5}$. This will have the effect of appearing to confirm those regulatory judgments, while relegating to a background role most of the valid alternative estimates that inconveniently fail to support EPA's regulatory judgments. This is at odds with the primary value of risk analyses, which is to provide understanding of the implications of the current state of knowledge to help form such regulatory judgments. It is also at odds with recent guidelines from the Office of Management and Budget (OMB) for ensuring that Federal agencies disseminate objective and useful information.

I have a number of other concerns on the Plan that I also summarize in these comments. These relate to its vagueness about what calculations EPA actually will perform, its lack of focus on critical issues that should be given a central role in a balanced risk analysis, and some apparent errors in the document.

THE PROPOSED APPROACH IS INCONSISTENT WITH THE STATED ANALYSIS GOALS

The core issue in $PM_{2.5}$ risk analysis is the sheer scientific uncertainty surrounding any single risk estimate. The Plan discusses uncertainty, but does not offer a methodology for addressing these uncertainties quantitatively. Rather, the Plan uses uncertainties as an

excuse not to use the resulting risk estimates to help select among alternative standard levels:

> *"EPA recognizes that the role of the risk analyses in this standards review will necessarily be limited by significant uncertainties … and does not plan to use the risk estimates as a basis for recommending selection among alternative standard levels."* [1]

Limiting the use of the risk estimates in this way is not an appropriate response to the presence of large uncertainties. The appropriate analytical response is to integrate the most sensitive of the uncertainties into the quantitative analysis. With uncertainties properly reflected in the analysis, the risk analysis could finally become useful in helping setting the level of the standard.

In place of its role in helping inform the standard setting process, EPA states:

> *"The goals of the Proposed PM risk analyses are: (1) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates and (2) to gain qualitative insights into the nature of the risks associated with exposures to PM."*[2]

However, the Plan fails to provide for a complete reflection of the current state of knowledge, and in particular, of how all possible alternative assumptions affect the risk estimates. There is, in particular, a rigid adherence to EPA's regulatory position that all PM constituents can be treated as equally potent and therefore that risks of $PM_{2.5}$ can be reduced effectively by regulating them with a generic mass-based ambient standard. Insistence on using only this assumption is jarring because the Plan acknowledges repeatedly that there is enormous uncertainty regarding which components in the $PM_{2.5}$ mix might be most potent. Inconsistent with the goals quoted above, the planned risk analysis would make no attempt to "develop a better understanding of the influence" on risk estimates of this assumption regarding relative constituent potency; it would make no attempt to gain "qualitative insights" into the nature of this major source of uncertainty.

There is no excuse for efforts to brush aside some of the most critical uncertainties associated with regulation of $PM_{2.5}$, especially given that EPA "does not plan to use the risk estimates as a basis for recommending selection among alternative standard levels." In fact, EPA would be creating a biased impression in favor of *any* standard that it may recommend if it simultaneously releases a near-deterministic risk analysis that is based on assumptions that do not reflect the current state of uncertainty and which are instead selected implicitly because they will support any standard down to background levels.

In January, the OMB published its "Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by Federal Agencies". This document states:

[1] Plan at 3.
[2] Plan at 3.

> *" 'Utility' refers to the usefulness of the information to its intended users, including the public. In assessing the usefulness of information that the agency disseminates to the public, the agency needs to consider the uses of the information not only from the perspective of the agency but also from the perspective of the public."* [3]

EPA's risk analysis plan is inconsistent with these guidelines because it will not provide information that has usefulness to either the EPA or to the public to which it will be disseminated. If EPA really does not intend to use the results of the proposed risk analysis to help guide its choice of $PM_{2.5}$ standard, then one must wonder why the risk analysis is being done at all. If, however, the ambient standard *were* to be supported by a risk analysis, then the only way that the risk analysis could have any utility to the public or to EPA would be for it to objectively quantify the implications of the current uncertainties for potential risk reductions. If EPA does not modify its plan to include a more complete and unbiased representation of the current uncertainties, then the PM risk analysis will violate the OMB Guidelines for maximizing usefulness and objectivity of the information that Federal agencies disseminate.

In short, having no risk estimates at all would be better than having risk estimates that are neither designed to provide understanding of uncertainties, nor designed to help set the $PM_{2.5}$ standard.

The most important attribute of an analysis that is intended to provide understanding of the role of inputs and assumptions is that it not prematurely eliminate representation and consideration of key sources of information, even if these sources are at early stages of research or difficult to quantify. Several specific omissions in the Plan will severely limit the ability of EPA's risk analyses to improve understanding of the impact of different assumptions. These omissions need to be rectified in order for the risk analysis to meet the goals stated in the Plan. Specifically:

- The Plan should provide for a full and open exploration of the difficulties in identifying the relative toxicity of $PM_{2.5}$ constituents. The risk analysis should be designed to illuminate how this significant unknown may affect the estimates of risk under a "rolled back" level of $PM_{2.5}$ mass (and hence will affect the estimates of risk *reduction* associated with controlling $PM_{2.5}$). At present, this absolutely overarching source of uncertainty is not even listed in Exhibit 2.5 as a proposed sensitivity analysis. This is remarkable given that OMB specifically highlighted it in a letter to Administrator Whitman as one of the top "critical research needs that can help target environmental-protection investments to the most important sources of PM." [4] This uncertainty requires more analytical emphasis than is provided by sensitivity analyses alone, which can only confirm that it is a critical uncertainty.

[3] OMB (2002), §V.2.
[4] Graham (2001), p.1.

- Of particular interest to the assessment of constituent-level relative toxicity would be information available in the single epidemiological study that has actually been performed using monitoring data that separates the monitored $PM_{2.5}$ mass into its key components. That study, by Klemm and Mason (2000), is not even listed for consideration in the current Plan (see Exhibit C.1). The ostensible reason for its omission is that it does not appear in a particular summary table in the PM Criteria Document (PM CD). However, an uncertainty analysis should not slavishly adhere to the PM CD's summary table, which was prepared for a totally different use, to reflect the body of completed research. EPA's goal for the risk analysis is an exploration of that which is not yet known, and this goal cannot be met by adhering only to research that the PM CD deems complete. OMB also noted the importance of this "Atlanta study" in its letter to Administrator Whitman.[5]

- The Plan does not recognize the significant uncertainties that have been highlighted in the chronic mortality literature since 1997. One important new piece of research is the Veteran's Cohort Study by Lipfert et al. (2000). The Plan omits all reference to this study, which sheds some interesting new light on this rather limited area of epidemiological findings. A justification for its omission from the list of candidate chronic mortality studies in Exhibit C.4 is quite disingenuous: "Exhibit C.4 presents a summary of …studies identified in the draft PM CD…that report effects estimates for long-term exposure mortality associated with $PM_{2.5}$."[6] This sounds as if the exhibit lists all the long-term mortality studies that are discussed in the PM CD. However, it is really saying that the only chronic mortality studies that EPA would consider in its risk analysis are those that find a positive and significant effect on mortality. Lipfert et al. does not end up on the list for the simple reason that it does *not* find a $PM_{2.5}$-mortality relationship (even though it does find interesting and coherent mortality associations for other pollutants such as ozone). An objective assessment of the full range of relevant risk information cannot exclude one of only three peer-reviewed studies simply because it comes up with a result opposite from the other two. Lipfert et al. should be added to the list in Exhibit C.4.

- A second new area of uncertainty that has emerged in the chronic mortality literature relates to problems that Krewski et al. (2000) have identified regarding the statistical properties in analyses using the two data sets that are cited in the Plan: the ACS and Six-Cities data. EPA rightfully recommends using only the Krewski et al. (2000) reanalyses of these two data sets. However, the relative risk data cited in Exhibit C.4 imply that EPA intends to use only the original regression formulations out of the Krewski et al. study. In fact, the authors explored a wide range of alternative specifications, and identified a fundamental flaw in the statistical properties of the original formulations, spatial autocorrelation. After quite thorough attempts to understand and control for this spatial autocorrelation, Krewski et al. arrived at relative risks for $PM_{2.5}$ that were much smaller than those reported in Exhibit C.4 and also were found to be statistically indistinguishable from no risk at all (i.e., "insignificant"). There is no acknowledgment of this issue in EPA's Plan, let alone

[5] Graham (2001), p.2.
[6] Plan at C-3.

an explanation of how EPA intends to address this key issue. An objective analysis would start from regressions that have more credible statistical properties, and would certainly make an effort to directly address the attending statistical uncertainties. These are significant omissions in EPA's Plan.

- EPA suggests that it will continue to present "confidence intervals" that would be derived solely from the statistical standard errors on individual regression coefficients in a single study.[7] The standard errors in a single study help one interpret the robustness of any relationship identified in that study. However, in light of all the other large sources of uncertainty,[8] presentation of risk estimate ranges based on statistical errors alone would be highly misleading, even if accompanied by the caveats that EPA has inserted into the Plan.[9]

THE PLAN IS TOO VAGUE FOR A FULLY INFORMED COMMENTARY

The Plan is far too vague about what it specifically intends to do. It provides a summary of the standard concentration-response (C-R) functional form, and it provides extensive discussion of "roll-back" methods for simulating the air quality implications of alternative standards. However, almost all other aspects of the analysis (which are the ones that will most affect the quality of that analysis) are described only as "similar to the methods used in the previous PM risk analyses."[10] Without providing this specificity now, EPA cannot later claim that the public had an ample opportunity to comment on its actual risk analysis methods.

If the Plan is to simply repeat the techniques of the last iteration, then there are substantial problems with this Plan, as were documented in my comments at the time of the previous PM risk analysis in 1996-1997. These criticisms include:

- Inappropriate use of statistical standard errors as the sole indicator of uncertainty in point estimates of risk taken from a single study.

- Failure to recognize the key role of the assumptions that there are no thresholds or other significant non-linearities in the C-R functions, particularly at the lower range of concentrations.

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[7] See Plan at 23.

[8] The other important uncertainty issues specifically include: variations in the potencies of the different $PM_{2.5}$ constituent; the inability of statistical methods applied to these types of data to identify functional forms such as the presence of a threshold; the potential causal role of co-pollutants; the wide range of different findings across many studies; variability in the findings for different regressions even within one study; and the evidence that classical statistical properties may not even hold in some of these studies (e.g. due to spatial autocorrelation).

[9] EPA states in the Plan that statistically-derived "confidence intervals will express the range within which the true risks are likely to fall *if the uncertainty surrounding PM coefficient estimates were the only uncertainty in the analysis*." (Plan at 23). Since statistical variability is <u>not</u> the only source of uncertainty, an uncertainty range based on statistical uncertainty alone is de facto <u>not</u> useful or objective information to disseminate.

[10] Plan at 4.

- Failure to incorporate the full range of relative risk values from multiple studies.

- Failure to address uncertainties associated with differential constituent potencies, especially in light of the fact that control strategies will almost certainly roll back some constituents more than others.

- Failure to address the possibility that correlations among pollutants, when combined with the fact that exposures to different pollutants are estimated with different amounts of error, may result in false but statistically significant positive associations of health impacts with a non-culprit pollutant.

- Failure to give sufficient emphasis in the exposition of results to the full range of uncertainty represented in integrated sensitivity scenarios. (The analysis of risks should be centered around an integrated representation of uncertainties, and this type of analysis should not be relegated to the back pages of a document or some technical appendix.)

To this earlier set of criticisms, I now add another, because of the unstated but implied possibility that EPA may be intending to combine multiple mortality studies into a single C-R function:

- Use of Monte Carlo analysis is an inappropriate way of combining results from multiple studies when the statistical properties of the standard errors are in doubt, such as when there are known biases due to spatial autocorrelation.

Another concern regarding the *implied* methodology relates to the use of single vs. multi-pollutant regressions. EPA does not commit specifically to using one or the other, but the entry in row 6 of Exhibit 2.5 suggests that EPA is intending to use a single-pollutant formulation and then to merely reflect the multi-pollutant option as a sensitivity case. There is no sound statistical argument for using a single pollutant regression as a base case. Reliance on single pollutant regressions reflects nothing more than a preference for assuming the largest possible relative risk regardless of potential statistical biases induced by failing to control for other possible explanatory factors. An interest in having an unbiased estimate of a $PM_{2.5}$ effect would dictate using the multi-pollutant formulation whenever it is available. At a minimum, for any study that offers both types of estimates, there should be an equal representation of the results using both formulations, and giving them equal weight in presentation and communication of results.

The Plan also needs to be much more specific about how EPA intends to address any of uncertainties in its quantitative estimates. For example, Section 2.8 titled "Characterizing uncertainty" merely states the "The following will be among the major sources of uncertainty in the risk analyses:…" and then proceeds to list some of the main forms of uncertainty.[11] However, the section never expressly commits to quantify any of the

[11] Plan at 22-23.

uncertainties except the statistical uncertainty.[12] Instead, the Plan vaguely suggests that additional uncertainties "*could* be included in a Monte Carlos analysis"[13], and that "*Possible* additional or alternative approaches to characterizing uncertainty that *are being considered* include the following: "integrated sensitivity analyses" *may* be presented….Different sets of plausible assumptions…*could* be presented. [emphases added]"[14]

Another striking example of vagueness is found in Section 4 on "Selecting Concentration-Response Functions". The Plan states:

> *"[S]tudies often report more than one estimated C-R function for the same location and health endpoint. Sometimes models including different sets of co-pollutants are estimated in a study; sometimes different lags are estimated. It is also possible that two different studies estimate a C-R function for the same combination of PM and health endpoint in the same location. It is therefore necessary to make decisions about which C-R functions to use in the risk analysis." [emphasis added]* [15]

The rest of the section, however, suggests a specific analytical decision for only one of the foregoing matters, that of lags. For all the others, the Plan proposes to report risk estimates based on all the available options. The Plan does not provide any insight on how EPA intends to do this.

- Do they propose to combine different types of risk estimates using some technique that is not mentioned?

- Do they instead propose to literally report risk estimates based on all the various combinations of these alternatives?

- Do they intend to pick a preferred option (which has not been identified yet) and then only apply the other options in sensitivity analyses?

The most likely outcome (to a person trying to read between the lines of the Plan) seems to be the third of the above possibilities: it would be "similar to the methods used in the previous PM risk analyses"[16] and it would be consistent with the way the discussion of sensitivity analyses is set up in Exhibit 2.5. The third approach would be a concern for two reasons:

(1) It sets up a situation where a single point-estimate of risks will be provided in a deterministic manner that masks the true sense of uncertainties. Any sense of

[12] See my comments above that make it clear that it is misleading and inappropriate to quantify *only* the statistical uncertainty while not quantifying any of the much larger sources of true uncertainty in the $PM_{2.5}$ risk relationship.
[13] Plan at 23.
[14] Plan at 23-23.
[15] Plan at 32.
[16] Plan at 4.

uncertainties would be relegated to sensitivity analyses that only the technically-inclined public would read.

(2) EPA has not made clear what it will choose to assume for the point estimates of risk. This leaves the public with no opportunity to comment on some of the most crucial decisions of a deterministic risk analysis.

In summary, it is not sufficient for the Plan to say that where two alternative assumptions are available, "risk reduction estimates based on both will be reported."[17] It is important for the Plan to be revised to explain how EPA intends to provide balance in the way that these multiple risk estimates are to be communicated in the resulting report. It is important for the Plan also to explain how multiple different sources of uncertainty will be integrated together into a unified representation of uncertainty that will replace the misleading use of quantitative uncertainty bounds based solely on statistical errors in the studies.

HEALTH EFFECTS STUDIES ARE BEING ELIMINATED FROM CONSIDERATION ON AN ARBITRARY AND UNNECESSARY BASIS

The Plan proposes to screen out any acute effects epidemiological study that has a "natural log of mortality-days" that is less than 9.0. This metric is a completely new construct that has no foundation in the statistics literature. The Plan suggests that the justification for this screening rule is in the March 2001 draft PM CD. However, a review of the relevant portions of the PM CD makes it clear that this metric was devised as a method for attempting to explain the heterogeneity found in the PM_{10} acute mortality studies of Samet et al. (2000).[18] The metric was *not* used as a screening device for comparing across studies using very different methodologies. It is quite possible that a study of very high methodological quality may provide more powerful risk estimates than a study with a larger number of mortality-days but weaker methodological techniques. The metric proposed by EPA is therefore being used in an inappropriate manner to identify "good studies", and this is inconsistent with its original use. Further, the cut-off point of 9.0 is arbitrary. A review of Exhibit C.1 suggests that this bright-line approach is causing some studies to be eliminated from consideration over relatively minor differences in the size of their data sets.

Design of the study is what determines whether a particular statistical standard error measure is trustworthy for purposes of inference about confidence intervals. A better screening device therefore would consider the relative technical merits of the various studies, the quality of the data used in the study, and what types of information a study can provide to help develop a better understanding of the qualitative nature of risks. These considerations should be given at least as much weight as the simplistic metric of numbers of mortality-days in the data base.

[17] Plan at 34.
[18] PM CD, March 2001, p. 6-260.

Most importantly, the risk analysis should not screen out studies for the mere sake of limiting the universe of options down to just one or two studies. Once a particular health effect and location are identified for analysis, a good risk analysis should strive to incorporate all of the information from all of the applicable studies. Different weights may be assigned to different studies if some are deemed to have stronger overall technical merits than others, but there is no reason to screen out certain studies on the mere basis of having fewer data points than other available studies.

For the same reasons, the Plan should not rely just on the list of studies from the Summary Chapter of the PM CD (i.e., Table 9-3 of the PM CD). This table does not reflect all of the relevant new studies that are described in the Epidemiology Chapter (Chapter 6 of the PM CD), and it is unclear what judgments were made in screening out studies that were discussed in the Epidemiology Chapter.[19] Further, the goal of a sound risk analysis is to reflect all of the relevant information that relates to estimating risks. This may require use of emerging research when no comparable form of research has yet been completed. Emerging new information that can significantly alter estimates of risk is always relevant to an uncertainty analysis, even if given less weight when integrating uncertainties.

THE PLAN LACKS FOCUS ON WHAT MOST MATTERS FOR PRODUCING SOUND RISK ESTIMATES

Throughout the document there is no coherent plan for addressing uncertainty in a manageable way. Minor sources of error are given greater attention in the Plan than the truly significant sources of uncertainty. The only way to move towards a risk analysis that provides "a better understanding of the influence of various inputs and assumptions on the risk estimates" and offers readers balanced and objective "qualitative insights into the nature of the risks"[20] is to prioritize the sources of uncertainty. The analysis must focus only on a few sources of uncertainty that do have the potential to dramatically change the sense of the risks. Sensitivity analyses are intended to aid in this process of focusing the uncertainty analysis. EPA and others have had enough previous experience with PM risk analyses to know that the critical driving sources of uncertainty (excluding the presumption of causality itself) are:

(1) The likelihood that potency is concentrated in only one or a few of the many $PM_{2.5}$ constituents that may be the target of a control strategy.

(2) The potential that the true dose-response relationship is highly non-linear at concentrations of $PM_{2.5}$ above background levels (e.g., an effects "threshold" may exist).

[19] Examples of studies in Chapter 6 of the PM CD that would be very informative to a risk analysis, but which would be ignored under EPA's approach of using only studies cited in Chapter 9 of the PM CD include Klemm and Mason (2000), Lipfert et al. (2000), and Smith et al. (2000).
[20] Plan at 3.

(3) Consideration of regression models that provide control for statistically significant ecological covariates such as socioeconomic factors and co-pollutants, and which control for spatial autocorrelation.

All of the rest of the many sources of uncertainty and imprecision described in the Plan are minor compared to these. All 12 pages of Section 6 of the Plan on "Sources of Uncertainty" could be removed without loss of insights on the matter of uncertainty, if it were to be replaced by a coherent Plan for addressing the above three issues.

In contrast, Section 6 states that the issue of relative potency will be omitted altogether from any discussion of uncertainty: "the chemical composition of PM will not be considered explicitly in any of the risk analyses."[21, 22]

At present, Section 6 does suggest it will address the question of a threshold, but it provides no clear plan to address this uncertainty other than some sensitivity analyses of unspecified scope. As noted above, sensitivity analyses are insufficient for integrating these uncertainties into a complete and balanced representation of the range of potential risks from $PM_{2.5}$. Sensitivity analysis is just a screening tool for identifying uncertainties that need to be incorporated into an integrated risk analysis. We don't need any more sensitivity analyses here because the threshold question has already been identified in numerous previous analyses as a very critical source of uncertainty. Instead, EPA now needs to produce a plan that integrates this uncertainty into its risk analysis. In doing so, EPA needs to include studies that report evidence of thresholds, such as Smith et al. (2000).

Section 6 provides no plan for addressing the uncertainties posed by alternative levels of controlling for covariates and other statistical problems. It tries to dismiss this issue when it states that "[t]echniques for addressing the problem of confounding factors and other study design issues have improved over the years, however, and the epidemiological studies currently available for use in the PM risk analyses provide a higher level of confidence in study quality than ever before."[23] This statement may be true, but it does not indicate whether EPA intends to use the best-controlled studies and models now available. For example, the most thorough analysis of chronic mortality (by Krewski et al., 2000) offers dozens of increasingly well-controlled regressions. EPA does not indicate whether it will continue to use the less-controlled regressions from this study, or actually rely on the best-controlled regression out of that study. The outcome of

[21] Plan at 49.

[22] On p. 30, EPA states that geographical variation in the composition of ambient PM creates so much uncertainty that they prefer not to extrapolate risk estimates results to a national scale. This same problem is a dramatic source of uncertainty even for the risk analysis limited to specific urban areas. Even if the risk studies being used were estimated in the local area for which risk estimates are being made, any strategy to reduce PM will almost certainly change the composition of that area's ambient PM. Some constituents will be rolled back substantially more than others, and the uncertainty due to variation in PM composition cannot be avoided by limiting the analysis to just a few cities. Thus, variation in composition is not a reason to avoid a national scale analysis; rather, it is an uncertainty that needs to be addressed even if only one city were the subject of the risk analysis.

[23] Plan at 46.

this decision will produce an extremely different set of risk estimates.[24] Exhibit C.4 hints that EPA intends to use the less fully controlled regressions that produce higher risk estimates.

It should be noted that the roll-back methods, which receive special attention in the Plan, only affect impacts based on daily data. Since these are usually far smaller than those that result from chronic studies, there is relatively little value to having so much detail on such a minor point, while truly major methodological issues such as how manage the findings on spatial autocorrelation by Krewski et al. are not even mentioned in the Plan.

MISLEADING ASPECTS OF EXHIBIT 6.1

The most misleading aspect of Exhibit 6.1 "Key Uncertainties in the Risk Analyses"[25] is that it fails to identify *key* uncertainties. It randomly mixes many minor or almost irrelevant sources of variability in with the key sources of uncertainty that may cause true biases in risk analysis results. The table should be reconstructed so that it separates sources of potential bias from sources of random error, and so that it highlights the key uncertainties based on a six-year history of performing sensitivity analyses for $PM_{2.5}$ risk estimates.

As such restructuring of Exhibit 6.1 is being done, it should modify the current entries that are misleading or wrong:

- The presumption that there is a causal relationship between $PM_{2.5}$ and mortality is a major source of potential bias. However, at present this major issue has been artificially combined with the more mundane issue of statistical variability in empirically estimated C-R functions. The effect of this artificial combination is that EPA is able to report the potential direction of error associated with causality as being uncertain, even though it is clearly a major source of upward potential bias. It should be completely separated from the random errors associated with empirically estimated C-R coefficients, which do have an uncertain impact on the risk estimate (but a much smaller one).

- Lack of knowledge of true functional form in a C-R relation poses a clear upward bias in risk estimates for PM because these risk estimates will be for reductions, not increases in air concentrations. The table, however, suggests that the direction of error is unknown. When moving towards lower and lower concentrations, the primary difference in functional form from that assumed for the empirically estimated C-R functions will be the potential for a threshold (or other less dramatic form of non-linearity) that implies lower risk per unit of PM at the lower end of the exposure range. Thus, for risk assessments that consider the benefits of decreasing air concentrations from those currently being experienced, the direction of error is an

[24] The best-controlled regression in Krewski et al. produces a smaller (and statistically insignificant) relative risk than that which results for the original formulations.
[25] Plan at 43-45.

upward bias in risk estimates based on the linear forumulation of empirical C-R functions.

- Extrapolation of C-R relations beyond the range of observed PM data also creates a definite upward bias in the estimated changes in risk, given that the direction of changes in concentrations will always be towards the lower end of the range of observed PM data in the study. The methodological decision not to calculate risks below the lowest reported value in a study does not eliminate this bias, and does not alter the fact that the bias is definitely in the upward direction.

- "Adequacy of PM characterization" addresses the uncertainty regarding constituent potencies. The entry here claims that much of this uncertainty is mitigated because EPA will estimate risks for each city using C-R functions only from studies based in the same city. This statement complete misses the point about the uncertainty in risk reduction when we do not know which $PM_{2.5}$ constituents are potent. When air concentrations are reduced under the "rolled back" scenarios, EPA's risk analysis is completely uninformed about whether the potent constituents in that city are being rolled back, or primarily the non-potent constituents. Thus, the enormous uncertainty associated with $PM_{2.5}$ constituent potency remains in the estimates of risk under rolled-back conditions in a city, and therefore also in estimates of risk reduction associated with reduced air concentrations. The statement should be deleted.

- Some of the entries in Exhibit 6.1 apply only to daily forms of C-R relationships. This makes these sources of errors less important to the overall risk analysis which experience tells us will be dominated by the chronic mortality estimates. The relevance only to daily studies should be noted for "Lag structure of C-R relation," and "Adjustment of air quality distributions to simulate just meeting alternative standards."

INCONSISTENCIES IN EXHIBIT C.1

My comments above argue to eliminate the plan to use only studies from Exhibit C.1 that also meet the 9.0 ln(mortality-days) criterion. Additional studies may need to be brought onto the list. One important example is Klemm and Mason (2000) because this is the only available study that can shed any light on constituent-level potencies. Nevertheless, it is still important to point out that Exhibit C.1 appears to contain numerous inconsistencies between the supposed rules for screening and actual outcomes, as indicated by bold font. These are noted below. References to the studies listed below can be found in Exhibit C.1 of the Plan.

Some studies are above the 9.0 cut-off and located in cities that have sufficient air quality data, yet they are not shown in bold font. Is this an error, or was some other reason applied to screen these studies out? The examples are:

- Schwartz et al., 1996; Boston, MA (for mortality)
- Schwartz et al., 1996; Portage, WI (for mortality)

Some studies are below the 9.0 cut-off, yet they appear to be included anyway because they are shown in bold font. Is this an error, or was some other reason applied to let these studies be included despite their failure to meet the bright line cut-off? The examples are:

- Fairley, 1999; Santa Clara Co., CA (for total cardiovascular and total respiratory)
- Mar et al., 2000; Phoenix, AZ (for total cardiovascular)
- Lippmann et al., 2000; Detroit MI (for total respiratory, pneumonia, and COPD)
- Moolgavkar et al., 2000; King Co., WA (for ER visits)
- Moolgavkar et al., 2000; Los Angeles, CA (for ER visits)

Two studies are above the 9.0 cut-off but no air quality data was presented at all, so it is not clear if these cities had insufficient data or if there is another reason they are not shown in bold font:

- Schwartz et al., 1996; Knoxville, TN (for mortality)
- Tolbert et al. 2000a; Atlanta, GA (for ER visits)

Finally, the Plan does not give a good explanation for why it will use Schwartz and Neas (2000) for respiratory symptom endpoints without subjecting this study to the same screening process.

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Attachment G

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May 5, 2003

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Re: **CASAC Review of Preliminary Recommended Methodology for Coarse Particulate Risk Analysis**

Dear Dr. Hopke:

These comments are offered by the Coalition for Coarse Particle Regulation, with respect to the recent draft risk assessment methodology for coarse particulate matter (PMc).[1] The comments were prepared with the assistance of Drs. Jay Turim and William Pepelko of Sciences International, Inc., and Dr. John Richards of Air Control Techniques, Inc. Drs. Turim and Richards have addressed the Committee at past meetings on these issues.

During the May 1 meeting to discuss the draft methodology, several members of the Committee noted the "minimalistic" nature of the health effects and exposure data available for use in a risk assessment for PMc. Nevertheless, the Committee apparently intends to recommend that EPA proceed with assessments based on the PMc morbidity and mortality data.

In prior CASAC meetings, Drs. Turim and Richards have testified that the uncertainties in the PMc data are so great as to render them unable to produce any scientifically sound quantitative assessment of potential risk from PMc exposure. Their view is supported by the discussions of the data in the draft PM Criteria Documents issued to date, and the draft risk assessment methodology does not include any detailed assessment of the suitability of the chosen data for purposes of quantitative risk assessment.

[1] Abt Associates, "Preliminary Recommended Methodology for PM10 and PM 10-2.5 Risk Analyses in Light of Reanalyzed Study Results" (April 3 2003 Draft). The Coalition consists of the following organizations: National Mining Association, National Stone, Sand and Gravel Association, Industrial Minerals Association -North America, and American Forest and Paper Association.

We urge the Committee to reconsider its apparent decision to support quantitative risk assessment based on the extremely limited data available for PMc at this time. If the Committee believes that EPA should proceed with its assessment despite the numerous uncertainties in the underlying data, we ask the Committee to make it clear that EPA's analysis must include a detailed description of the uncertainties and their effects on the risk assessment results. These points are discussed in detail below.

Health Effects Data

Mortality

The Abt Associates draft states that "the risk analyses will be predicated on the assumption that the relationships are causal" (p. 2). However, we are not aware of any study that has drawn that conclusion, and it should not be assumed given the widespread uncertainties inherent in the current data. The final methodology should include a detailed discussion of the suitability of the chosen data for purposes of quantitative risk assessment, including the the level of uncertainty in the selected dose-response relationships. We are attaching the relevant discussions of the mortality and morbidity data from the most recent draft PM Criteria Document. Major uncertainties in the current mortality data include:

- The number of coarse particle studies is quite small compared to the overall PM data set, and the studies include all of the uncertainties inherent in reliance on ambient monitoring data;

- The results are not confirmed in chronic exposure studies;

- The short-term study results are mixed, and often inconsistent with the larger body of fine particle studies (see, e.g., the discussion of the Phoenix data);

- Only three of the eleven available studies reported statistically significant effects, and those correlations are generally weak and may be driven by temporally covarying fine particles (see the discussion of the Detroit data);

- The sample sizes generally are small and not sufficient for separation of small differences in reported effects;

- Many of the positive associations were observed during warmer seasons, suggesting dependence on biologically-derived particles (molds, endotoxins, etc.) elevated during such seasons. Yet the Abt report indicates that it is not possible to prepare a seasonal sensitivity analysis for the PMc data (p. 14);

- The studies generally involve urban areas with a wide variety of PM chemical compositions that are not representative of PMc throughout the U.S.,

particularly with respect to the varying content of toxic, biological and crustal materials.

Morbidity

With respect to the morbidity data as a whole , the draft CD concludes that "insufficient data exists from these relatively limited studies to allow strong conclusions at this time as to which size-related ambient PM components may be most strongly related to one or another mortality endpoints" (pp. 8-236-37). This is confirmed by a detailed examination of the PMc studies discussed in the Abt paper.

The draft CD includes very little discussion of the Schwartz and Neas paper on which the PMc assessment would be based. This paper reports the results of a two-part investigation of acute respiratory effects in children residing in Uniontown, PA and State College, PA. In the first part, children in grades 2-5 were requested to keep a diary recording occurrence of respiratory symptoms which were defined as any day with a report of coughing, phlegm, pain in chest or wheezing. Reported effects were related to air particle concentrations. The study found larger effects for PM2.5 than for PM10. A statistically significant, but very small response was noted for lower respiratory symptoms during exposure to increased PM2.5 but not PM10. A statistically significant increase was reported for cough during exposure to 8 µg/cu m coarse particles, but not 15 µg/cu m fine particles. The increase in cough is a questionable finding since even higher concentrations of fine particles did not induce a significant effect. Self reported symptoms in children of this age are also a very questionable set of data from which to draw any conclusions.

In the second part of the investigation peak expiratory flow rates were measured twice daily in unsupervised tests conducted by the children. In the Uniontown group increased coarse particle exposure resulted in an increase in peak flow, but in the State College group peak flow was decreased; neither result was statistically significant. For fine particles peak flow was significantly decreased at Uniontown, but not State College. These changes were very small, in opposite directions for the coarse and fine particles in the Uniontown children. Moreover, peak expiratory flow rate is a crude measure of pulmonary function. The limited data provided by this investigation do not appear to provide an adequate basis for comparing effects of coarse and fine particles.

The Lippmann et al. study which would also be used in the PMc assessment contains a similar set of uncertainties. As noted in the draft CD, the temporal correlation between TSP and PM2.5 suggests that "much of the apparent larger particle effects may well be driven by temporally covarying smaller PM2.5 particles" (p. 8-44). In addition, there was a very strong association with sulfates. Air pollution data were collected from the Detroit Windsor area. Exposures were compared with mortality data and hospital admissions of elderly people. The cohort consisted of Medicare patients 65 and older. Data were collected for emergency and urgent hospital admissions only. Relative risks for pneumonia, COPD, ischemic heart disease,

dysrythmias, heart failure, and stroke were tabulated for lag days 1, 2, and 3 during exposure to increased PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$. While time constraints prohibited a detailed evaluation of the data tables for purposes of these comments, it appears that it would be extremely difficult to assign degree of effect among the three particulate categories. Moreover, the authors studied an elderly population (over 65 years of age at time of admission), and the ones admitted were likely to be very sensitive cases. In setting ambient air quality standards, Congress has directed EPA to choose exposure levels that protect sensitive populations but not highly sensitive individuals. Additional information on the population studied in this report is needed before the study can be used as a potential basis for PM ambient standards.

Crustal Particles

If EPA proceeds with a risk assessment for coarse particles, the analysis should attempt to segregate particles of crustal origin. The draft CD makes it clear in several places that crustal particles are unlikely to cause adverse health effects under most ambient exposure conditions (e.g., pp. 8-47-48; 8-284).

Exposure Issues

Any risk assessment that EPA prepares for coarse particles should include a thorough discussion of the uncertainties inherent in the available exposure data. Chapter 5 of the draft CD continues to find that it is important to understand the personal PM exposures of the populations evaluated in the epidemiological studies. It also continues to recognize that exposures to indoor PM can be substantial. However, the recent epidemiological studies contain virtually no personal or indoor PM exposure data. Rather, the reported epidemiological results are based on data from community monitors of ambient PM data. As a result, it is impossible to know the actual PM exposures of the persons included in the epidemiological reports with any reasonable degree of certainty.

The draft methodology does not include any detailed analysis of whether the exposure data in the epidemiological studies is capable of providing a sufficient basis for sound assessment of quantitative risk on which to base ambient PMc concentration limits. As discussed in CD Chapter 2, there are numerous uncertainties in the ambient data that should be factored into the human exposure analysis as well. This is particularly true with respect to the data for coarse PM, yet there is virtually no discussion in the Abt paper of the available exposure information for the coarse particle fraction. Major uncertainties in exposure data used in the current PMc studies include:

- Significant quality assurance issues involving the measurement of PMc using collocated PM10 and PM 2.5 monitors;

- Significant spatial nonuniformity of PMc values in a given metropolitan area due to rapid deposition of PM on the upper end of the PMc size range;

- Significant regional and seasonal differences in the presence of toxic and/or biological materials on the surfaces of PMc;

- Incursion of PM 2.5 into the PMc size range during periods of high relative humidity.

The attached paper published by Ono et al. in the July 2000 issue of the AWMA Journal documents large differences in measured values of PM-10 that result depending on the type of sampler used.[2] In the Ono study, the Wedding sampler was considerably lower than the Anderson sampler and both of them were lower than the dichotomous sampler. Additionally, the Ono data show that the cut point of the Wedding is closer to 7 than 10 microns. Depending on which sampler was used in the various epidemiological studies, in which the coarse fraction was estimated by subtracting reference monitor data from PM-10, the study results may have been substantially affected. The Ono report concludes:

> This study shows that in the absence of volatile particles and in the presence of fugitive dust, a different systematic bias of up to 35% exists between samplers using dichot inlets and high-volume samplers, which may cause the Graseby and Wedding samplers to undermeasure PM 10 by up to 35% when the PM 10 is predominantly from coarse particulate sources. . . This could affect PM 10 nonattainment designations and particulate control strategies in many areas. Epidemiologic studies that use multi-site and multi-city monitor data with a mix of PM 10 sampler types should consider incorporating this coarse particle bias into their statistical analyses to properly compare ambient PM 10 and coarse particle exposure effects on an equal basis. *This study shows that the coarse particle concentrations (particles between 10 and 2.5 micrometers) cannot be determined by a simple concentration difference between a PM 10 high volume sampler and a PM 2.5 sampler* (emphasis added).

The last, highlighted statement is particularly important here, as the Abt paper apparently derives simulated PMc concentrations by using precisely the method that was shown to be inaccurate in Dr. Ono's study. We understand that studies at other locations confirm Dr. Ono's findings. Any risk assessment that EPA performs for coarse particles should provide a detailed evaluation of the results of the Ono study and similar studies, and the potential effects on the epidemiological and risk assessment results.

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[2] Ono, et al, "Systematic Biases in Measured PM 10 Values with U.S. Environmental Protection Agency-Approved Samplers at Owens Lake, California," *AWMA Journal* (July (2000).

Conclusion

Toward the end of the May 1 meeting, it was suggested that EPA should move forward with PMc risk assessments based on very minimal data because the purpose of the Staff Paper and associated risk assessments is to provide information to the Administrator. It was also suggested that the assessments are not to be used as the primary quantitative basis for the new standards, as there will be a very high degree of uncertainty in these analyses. They should be viewed as "semi-quantitative" analyses that may help EPA understand the implications of the data but are not rigorous quantitative analyses.

For the reasons stated above, we agree that risk assessment based on the available PMc data would be of very limited utility, and we ask the Committee to reconsider its apparent decision to support PMc risk assessment based on the data currently available. If the Committee recommends that EPA proceed with PMc risk assessment, we urge the Committee to state clearly in its letter to EPA that these analyses should not be considered capable of providing a sound quantitative basis for new standards given the limitations of the underlying data.

Finally, we greatly appreciate the time and the effort that the Committee members have expended in reviewing these issues in detail. As Congress and EPA have recognized, your independent review is essential to ensure that our national ambient air quality standards are based on sound scientific determinations to the maximum possible extent. We look forward to continuing to work with the Committee on these issues as they evolve. If you have any questions with respect to these comments or would like to discuss them further, please contact me as indicated above.

Sincerely,

Kurt E. Blase

Counsel for the Coalition for
Coarse Particle Regulation

Cc: H. Richmond (EPA)